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ILLUSTRATED CLINICAL LECTURES AND
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ON

TREATMENT, MEDICINE, SURGERY, NEUROLOGY, PÆDIATRICS, OBSTETRICS, GYNÆCOLOGY, ORTHOPÆDICS, PATHOLOGY, DERMATOLOGY, OPHTHALMOLOGY, OTOLOGY, RHINOLOGY, LARYNGOLOGY, HYGIENE, AND OTHER TOPICS OF INTEREST TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION THROUGHOUT THE WORLD

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Diagnosis and Treatment

PARATHYROID TREATMENT OF PARALYSIS AGITANS

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This paper will consider briefly (1) the symptoms and (2) causation of paralysis agitans; then, at length, (3) the parathyroid treatment, and, finally, (4) useful accessory therapeutic resources.

It is believed that in this way the article will be made most useful to those who chance to be relatively unfamiliar with the course, management, and current views of this curious and baffling disease.

SYMPTOMS

There are but few symptoms of paralysis agitans which may not be referred mediately or immediately to the primary and striking feature of the disease in all its stages, *increased muscular tone.* Postponing till later (p. 4) a discussion of the origin of this phenomenon, it may be remarked here that when the contractile movements are intermittent (clonic) we get the feature of the disease from which it has derived its familiar name of "shaking palsy." When the impulses to the muscles are constant (tonic) we get, instead of clonic movements, a pronounced and permanent rigidity, commonly called *paralysis agitans sine agitatione.* The latter is apt to be a graver and more rapidly progressive form of the malady, and appears in relatively younger patients. Sometimes the two conditions coexist in the same patient, or the total and permanent rigidity may be a later stage. I have had one patient (female, 60 years) who, while entirely motionless and rigid to the eye of the observer, said she could herself feel very distinctly a shaking of her abdominal muscles.

In a large number of patients seen in the last ten years I have noted only about 2 per cent. in whom the shaking or rigidity was not greatly relieved, or entirely absent, in sleep; so that relaxation

during sleep ordinarily may be taken as a diagnostic sign. When the muscular tremor is present, it is characteristic, and to the practitioner familiar with paralysis agitans one look at the shaking lip or hand or foot is enough. The tremor is slow (3 to 6 vibrations per second), is aggravated by embarrassment or excitement, and is only temporarily controlled by mental effort.

The excursions of the various affected parts of the body may vary from ¼ inch to 2 inches or more. The tremor starts, as a rule, in one extremity—in one thumb or forefinger, or in one great toe—and spreads thence in the course of months or years to adjacent groups of muscles in the same limb, and to other parts of the body. The thumb and fingers often simulate a voluntary and intelligently directed movement, like pill-rolling or cloth-smoothing. Sometimes the arm and leg on one side are almost simultaneously affected, producing a "hemiplegic" form of the disease, often entirely misinterpreted by inexperienced practitioners. The slow onset, the character of the tremor (when present), and the absence of persistently exaggerated tendon reflexes on the affected side all differentiate unilateral paralysis agitans from the athetotic movements following hemiplegia.

Besides the more usual tremor and rigidity of the arms and legs, the muscles of the face are quite frequently affected, particularly the muscles of the lips. Occasionally the tongue is vibrated forward and backward uncontrollably. Dr. S. Wachsmann, of the Montefiore Home, New York, showed me some years ago a male patient of 55 years who had acquired a marked protrusion of the front teeth above and below from year-long pushing movements of the tongue.

The muscles of the larynx are apt to suffer early, and the articulation of the patient becomes slow and labored, while his voice diminishes in volume and alters in quality.

The entire face loses its muscular mobility, and a mask-like expression ("Parkinson mask") is frequently noted. This may be an early sign, and is of diagnostic value.

All voluntary movements are retarded in time, and require a special effort of will. An elderly man in my care, who had been a furnace-man for many years, complained that he had to "give up his job" because he could not put a shovelful of coal into the fur-

Fig. 1.

Front view of M. R., age 39 years. The picture, taken March 12, 1911, shows very well the posture, the flexor contractures of forearm, characteristic facies, and dropsical lower extremities. See also Fig. 2. (My thanks are due to Dr. S. Wachsmann, of the Montefiore Home, New York, for his kind permission to photograph this patient.)

Fig. 2.

Same patient as in Fig. 1, the photograph being taken in June, 1912. The progress of the disease is shown between the given dates.

nace door without stopping two minutes each time to "think it over." The muscular actions of daily life—walking, getting in and out of bed, going to the toilet, sitting down, standing up, dressing and undressing, shaking hands—become increasingly difficult, and finally impossible, and the unfortunate patient is ultimately converted into a wooden image of human form, limbs and back being almost immovable except in the hands of a careful and patient *masseur.*

A common condition in walking is *propulsion* or *retropulsion;* the patient, after starting to walk forward or backward, continues the action at a progressively rapid rate and is often unable to stop.

The flexors of the trunk and limbs being generally stronger than the extensors, there is a characteristic tendency to flexor contractures, and the rigidly bent posture of all advanced cases results, the arms, hands, and fingers being usually placed as in the adjoining photographs (Figs. 1, 2, and 3). Extensor contractures are also mentioned in the text-books. I have not personally observed any such.

There is no great loss of power in the muscles in the early stages, but later on a marked *weakness* results, and the sensation of weakness is to the patient often more tormenting than the rigidity.

Muscular and fascial pain is common. It often resembles muscular rheumatism, but is more likely due, in my opinion, to physiological muscular exhaustion. It may be compared to the pain and stiffness resulting normally from prolonged and overtaxing muscular exertion. Painful muscular cramps are common; curiously enough, particularly common in the little toe, which often gives the patient great distress.

A still more tormenting symptom is *restlessness.* The patient must change his position in bed dozens of times a night, move in his chair dozens of times a day, and where he cannot do this for himself, the nurse must do it for him.

The rigidity of the pelvic and abdominal muscles (especially the sphincter ani) often complicates severely the intestinal constipation from which these patients generally suffer. Piles may become aggravated, and the stiffness of the muscles of the arms and legs frequently produces an obstinate dropsy, which is not associated with either cardiac or renal disease.

Vasomotor instability is a rather marked symptom; it produces

hot and cold "flashes," or a persistent sense of heat or cold in one limb, in one half of the body, or over the whole superficies. Even in a cold room, the patient at night cannot perhaps bear the warmth of a single sheet.

The secretions are not affected, except those of the sweat and the salivary glands. There is no doubt that salivation is a genuine sign of paralysis agitans, though the ordinary text-book accounts ignore it. Naturally, when the tongue, the cheeks, and the muscles of deglutition are unmanageable, the patient drools, but saliva often fills the mouth before this muscular incompetence has developed.

In respect of the nervous system, the mind remains clear, though the patient naturally suffers from mental depression. Previously neurasthenic and psychasthenic patients often suffer an access of these symptoms after paralysis agitans develops, perhaps taxing to the utmost the resources of the medical attendant, but I do not believe that such symptoms are part and parcel of the disease.

The knee-reflexes are not permanently altered, though they may be increased or diminished for a few days at a time. Ankle clonus is absent. The cutaneous reflexes are normal.

In the older patients arteriosclerosis is common, but not constant.

The course of the disease, untreated, is slowly progressive. The cases with tremor are less progressive than those with predominant or severe rigidity. The average duration of the disease is from three to twenty years. *Spontaneous remissions* are not uncommon, and the practitioner is, therefore, always justified in assuming a hopeful attitude in the presence of the individual patient.

ETIOLOGY

The sex of the patient is immaterial. As to age, cases under twenty-eight are almost unknown, and under forty, rare. Paralysis agitans is essentially a disease of advancing years. It is common in all classes of society. Many patients tell of an acute illness, a psychic trauma, or a fall as antedating their trouble, but the connection is not clear. Syphilis is not associated with it in 10 per cent. of the cases. Autopsies have failed to show any gross lesion of brain or cord to which the disease may be reasonably attributed. C. D. Camp's interesting work upon the voluntary muscles in paralysis agitans will be referred to later.

Fig 3,

Mrs. S. B., aged 55 years, showing "Parkinson mask," posture of body and hands, and slightly swollen hands and feet (Courtesy of Dr. S. Wachsmann, Montefiore Home, New York.)

No hypothesis of the causation of the disease has gained any wide credence, except that vaguely suggested by Victor Horsley in 1885,[1] and more clearly enunciated by Lundborg[2] in 1904, and by myself[3] independently in 1905. This hypothesis is that paralysis agitans is due to deficient secretion of the parathyroid glands. Lundborg has frankly confessed his opinion to be based upon conjecture only—a conjecture suggested by the physiological explosion following parathyroidectomy, of which I shall speak more at length below. I have myself not been content with conjecture only, but have in the past seven years collected clinical and experimental data which go far, in my opinion, towards actually establishing this etiological relation. However, scientific truth is not to be reached by violence of assertion, but by orderly array of facts. These I shall state below, and leave the reader to draw his own conclusions.

My views have been confirmed quite generally by scientists and practitioners in this country and abroad—at least by those who have sought access to all that I have written on the subject in the last few years. One of the new German neurological text-books is still presuming that my first short paper on the subject, written seven years ago, is my final expression. G. Roussy and J. Clunet[4] have taken the opposite position, that paralysis agitans results from a hypersecretion of the parathyroid gland. In support of this view, they offer (1) autopsy findings in four cases; (2) bad clinical results in three cases from administering parathyroid gland, and (3) the good effect of exposing the necks of patients (three cases) to the X-rays.

As regards the value of the autopsy findings, I comment elsewhere (p. 7). Until it be proved exactly what was administered to the patients as parathyroid gland, and that they did not receive too large a dose, the therapeutic results are also of dubious significance. The patients distinctly benefited by my own preparation now number hundreds. And as regards the exposure of the neck to the X-rays, the parathyroids are probably too deeply imbedded to be affected in any way by such a procedure. What the exposure actually did was probably to reduce the activity of the *thyroids*. It has been known for many years that the administration of thyroid gland to Parkinson patients is distinctly hurtful.

The *anatomy* of the parathyroids has been frequently written up, and need not detain us (5, 6, 7, 9, 20).

As regards the *physiology* of the parathyroids, it may be briefly noted that when the parathyroid glands are removed from a dog or other suitable animal a curious and characteristic train of symptoms follows—hurried breathing, tachycardia, profuse salivation, twitching and shivering of the voluntary muscles, rigidity, intermittent convulsions, rapid wasting, and death. These symptoms have been so widely confirmed by so many different observers that the few dissenting voices nowadays are in a hopeless minority. The literature of the subject is enormous. A fairly complete bibliography may be compiled from the writings of Jeandelise,[5] Pool,[6] Erdheim,[7] Beebe and Berkeley,[8] and Ochsner and Thompson.[9]

MacCallum,[10] in a further interesting contribution to the subject, has shown that a suitable dose of a soluble calcium salt, injected into the veins of a dog that has been successfully operated upon in this way, will arrest the spasms and for a few hours restore the animal to a normal condition. He concludes that the parathyroid glands therefore preside in some way over the calcium metabolism of the body, and that the symptoms in question are caused by a deficiency of calcium. S. P. Beebe and I have successfully repeated this experiment, which is one of the most striking phenomena in the physiological laboratory; from a series of additional experiments, however, we have been disposed to conclude that in such cases the calcium has a "drug effect," and that the parathyroid glands more likely furnish enzymes of prime importance in the intermediary metabolism of nitrogen. The subject is a difficult one and awaits further research.

I have already remarked in another paper [21] that "Whatever the ultimate solution of the physiological problem may be, such remarkable and characteristic symptoms strongly suggest some causal relation between a defective secretion of these glands, and one or more of the convulsive diseases; and medical men have not been backward in guesses of various kinds. Exophthalmic goitre was long ago proposed by Gley [11] and afterward given over. Vassale proposed puerperal eclampsia.[12] Epilepsy was first suggested by MacCallum (acknowledgedly [13] in a tentative way), but further independent researches by this author [14] brought him to the conclusion (reached about the same time by Pineles [15] and Erdheim (*l. c.*), and rather vaguely suggested by Jeandelise (*l. c.*) in 1902, four years previously) that *tetany* is the final answer to this long-standing question."

Without going further into the extensive literature of this part of my topic, it will be enough to say that tetany—whether associated with surgical damage to the parathyroid glands in thyroidectomy, with infantile rickets, with pregnancy, or with dilation of the stomach—is unquestionably *one* of the answers. But a further inquiry remains whether this be the *only disease* having any relation to these glands, and it is in reply to this further question that Lundborg and I have suggested paralysis agitans as due to a disease or chronic chemical disorder of the parathyroid glands producing a deficiency of secretion.

In support of this hypothesis, I have offered the following considerations:

Paralysis agitans has all the marks of a chronic chemical poisoning. This view of the etiology of the disease has been taken for many years past by thoughtful and well-informed observers, and has been confirmed by the constantly negative character of autopsy findings. The "group-character" of the early tremors indicates, in my own opinion, that the primary site of damage is the motor centres of the cerebral cortex; the accuracy or inaccuracy of this deduction, however, does not affect the gland hypothesis. C. D. Camp,[16] in a laborious and interesting study, has collected the microscopic appearances of the muscles in a number of cases, and believes he has found a uniform lesion of the muscle-bundles. This awaits further confirmation; but whether nerve or muscle be the primary site, the glandular theory still holds.

Again, the symptoms following parathyroidectomy are remarkably like those of paralysis agitans. In certain stages of illness a parathyroidectomized rabbit has propulsion, tremors, rigidity, and salivation as perfectly as these signs ever appear in Parkinson's disease. If a chronic onset of parathyroid insufficiency could be induced in the laboratory, the resemblance might be yet closer.

Thirdly, in exophthalmic goitre and in myxœdema paralysis agitans has not infrequently occurred as a complication or sequela; and it is by no means too wild a suggestion that the diseased thyroid has damaged the adjacent glands by contiguity of tissue.

Fourthly, as regards autopsies, it is now possible to collate nineteen recent autopsies, carefully made upon cases of Parkinson's disease, in which the parathyroids were objects of special research.

Of these, nine have been reported by R. L. Thompson,[17] two by C. D. Camp (*l. c.*), one by myself,[18] two by Erdheim (*l. c.*), four by G. Roussy (*l. c.*), and one by Alquier.[19]

Alquier's case is imperfectly described, but the author evidently thought the gland in a diseased state. Erdheim's cases were negative. Thompson has published an elaborate and careful description of his cases, and concludes that "there is no morphological ground for the assumption that the parathyroid glands are insufficient in paralysis agitans." My own case, on the contrary, showed a definite degree of destructive sclerosis, but whether enough really to embarrass the function of the glands, it would be impossible to say. Of the two cases reported by Dr. Camp no elaborate description is given. The author's statement, however, is definite enough:

"I was able to examine the parathyroid glands from two of my patients, and in the opinion of Dr. Allen G. Smith, professor of pathology at the University of Pennsylvania, they were both in a distinctly pathological condition. In one there was some colloid material; . . . in both there was a peculiar infiltration with fat, especially in relation to the blood-vessels."

He concludes:

"This pathological evidence of disease of the parathyroids in paralysis agitans, when considered together with the experimental evidence and the therapeutical results of Berkeley, suggests that the parathyroid glands play an important part in the pathogenesis of paralysis agitans."

G. Roussy (*l. c.*) has reported four autopsies upon paralysis agitans. He gives carefully-made colored plates of the microscopic sections of the parathyroids, and describes them not as atrophic but as *hypertrophic*. The drawings are well done and interesting, but they do not appear to me at all convincing; the interpretation of them in any such way would be entirely subjective. I cannot do better than quote here what I wrote on the same subject[21] some years ago:

"Critical comment on so few cases would, of course, be provisional. In the light of these conflicting opinions no definite conclusions could be drawn. We must also note that the histology of the glands of internal secretion generally gives only a very elementary notion of their function. For example, from undoubted cases of Graves's disease thyroids are still sometimes taken in which the

histology is quite negative as regards the hyperplastic changes ordinarily found. Any one will recall, also, the extraordinary destruction of hepatic tissue occurring in the fatty degeneration of phthisis, or the malignant infiltration of secondary cancer. Yet the autopsy table is sometimes the first place where such changes are discovered, symptoms *intra vitam* being entirely negative. In respect of the parathyroids, we must further note that fatty infiltration and increased interlobular connective tissue are everyday appearances; and, indeed, the histology of the gland varies so greatly within seemingly normal limits that it would be safe to question the diagnosis of any pathologist who has examined the glands from less than seventy-five autopsies. This part of the subject must therefore be left till more reports are in."

We finally take up the *clinical facts.* And first I would note that a properly-identified gland must be used. Much of the commercial material can be given by the tablespoonful without damage or help. The only glands available for practical use are human glands gotten at autopsy, and ox glands. Horse gland is too expensive, rabbit gland and sheep gland are too small. Certainly for a long time to come experiments will have to be made with ox gland. Apparently bits of lymph node, thymus, accessory thyroid, and fat have done duty in many laborious experiments. Two German writers have recently announced some work upon tetany with "parathyroid glands." They gave bullock's or sheep's *thyroid* minced up with the adjacent fat—in the charitable hope that some of the parathyroid glands *might* be included somewhere in the mixture. The larger ox parathyroid lies in the upper end of the thymus gland near the cornu of the hyoid bone, and can be found, after sufficient instruction and practice, by any butcher who is handy with the knife and has good eyes.

A second point is that nothing much in the way of favorable results can be hoped for except from relatively small doses continued through a long period of time. Some patients have begun to respond in a week; some have shown no effects, good or bad, in less than six weeks. In some cases three months of patient and careful administration will have to be allowed before any conclusion, good or bad, is drawn. The extremely chronic way in which the disease

approaches would seem, indeed, to indicate that *slow regression* of the bad symptoms should be the rule rather than the exception.

A third point is that the temperament of these patients is peculiarly unstable. They are easily depressed, easily encouraged, given to rapid changes of opinion and mood, and very easily subject to suggestion. It is, therefore, positively essential to eliminate these factors before drawing a conclusion.

The clinical benefit has consisted in diminished rigidity, lessened pain, salivation cured, shaking diminished or cured, voluntary control of the muscles greatly increased, and restlessness and insomnia nearly or quite abolished.

The early cases in younger patients have responded with more speed and completeness, but unfortunate bedridden patients who had suffered for fifteen years with the disease have been quite often remarkably helped. One man of fifty-five years, with a rapidly progressive type of the disease, bedridden for six months, restless, sleepless, and greatly discouraged, was able to sleep again in three weeks, and for nearly a year returned to his work, going downtown every morning on the trolley alone.

Of another patient, who had been ill of the disease for some years, his wife wrote six months after treatment was begun: "He is improved beyond our most sanguine expectations. . . . He is in excellent spirits, and, in fact, I really expect that he will yet be entirely well."

Of a third patient, an elderly man who had been ill of paralysis agitans for many years and suffered much, his daughter wrote: "I should dread to contemplate my father's declining years if he had to get on again, as formerly, without your medicine."

The percentage of benefited cases in the last six years has increased *pari passu* with the gradual improvement I have succeeded in making my parathyroid extracts (p. 11). The number of patients happily affected is now well up in the hundreds, and over 80 per cent. of those who have tried my latest preparation have liked it and kept on with it. The dealers inform me that my new formula is not only popular in the United States, but that it is going abroad in large quantities, and that orders continue to come back from the same patients.

I have had but one or two patients out of the hundred and more

in my own care who really had an idiosyncrasy for ox-parathyroid. These could not take it in any form or dose without such disagreeable by-effects—"nervousness" and insomnia—that it was impracticable to continue the medication.

With such a percentage of benefited cases as above described, there seems to me no longer any reasonable doubt of the etiological relation between the disease and the remedy. One or two cases have appeared to be temporarily cured, but of such a disease, in a patient of advancing years, a "cure" is always to be mentioned with reserve.

I have ranged the whole field of internal secretions in the hope of reënforcing parathyroid by some other gland—on the hypothesis, now so popular, that all the internal secretions are intimately correlated. My results have been as follows:

Pituitary	No effect
Thyroid	Distinctly harmful
Pancreas (Fairchild's "holadin")	Harmful
Adrenal (Armour's)	Harmful
Prosecretin (Fairchild)	Harmful
Enterokinase (Fairchild)	Harmful

Finally, last winter, while working with Dr. C. L. Dana on pineal gland, it occurred to me to try pineal gland in combination with parathyroid upon some of my cases of shaking palsy. In three cases, so far tried, this combination has produced relatively happy results, but pineal gland alone has produced no effect that I have so far been able to notice.

PRACTICAL THERAPEUTICS WITH PARATHYROID

I come now to the practical administration of parathyroid glands to the patient.

My first cases were treated with commercial parathyroid gland. This worked well in a few cases, but was found so variable and uncertain in its action that it had to be given up. I next tried a preparation of properly-identified glands rubbed up in the crude fresh state to a dry powder with milk sugar. This was distinctly better, but still seemed to be variable and unstable. Later on I tried a nucleoproteid solution of the parathyroid made up by Beebe's method (*l. c.*) and preserved with a few drops of chloroform in the bottom

of each bottle. This has still further raised the percentage and degree of improvement, but it precipitates rapidly, and is very susceptible to damage from temperatures above 5° C., therefore hardly admitting of transportation to any great distance.

My latest process, devised over a year ago, and giving great satisfaction to a number of patients who had not done well on other preparations, is this: The nucleoproteid extraction process (Beebe's method, *l. c.*) is adhered to generally, but all the preliminary steps are hurried, and the precipitated nucleoproteid is not redissolved, but quickly dried in a draught of cold air; so that within eight or ten hours after the warm glands leave the bullock a minute amount of smooth, yellow powder is obtained which stands physiological tests admirably, is stable, easily handled, and does not require the ice-box for its preservation.

The powder is rubbed up with milk-sugar as a menstruum, and is dispensed in sealed and dated bottles of eighty capsules. Each capsule contains one-fiftieth of a grain of parathyroid nucleoproteid. The average dose is two per day—sometimes less than this where the patient is sensitive to the action of the remedy. This preparation is the best I have so far tried. It will give satisfaction not only in Parkinson's disease, but also in any form of tetany where the stomach can retain food. Otherwise, a sterile hypodermic preparation of the nucleoproteid must be used.

In paralysis agitans there are no special contra-indications, though some patients complain of "nervousness," inability to sit still, and an aggravation of their habitual constipation after a few days' dosage. Nothing is necessary in these cases except to diminish the dose and increase again more slowly. Little benefit can be expected of a satisfactory character except from small doses continued over a long period of time—three months to a year.

Patients taking parathyroid require to make no change in their mode of life, diet, or habits not otherwise indicated. Those able to attend to business should be encouraged to work, but with shorter hours, and more frequent periods of repose.

There are a few patients now and then who do much better on the above-named hypodermic preparation than on capsules. The hypodermic solution of nucleoproteid is easily administered, and causes little or no pain or irritation, if given with proper aseptic precau-

tions. Sometimes a capsule may be given in the morning and a hypodermic injection at bedtime—thus securing for the patient a restful night. The base of the solution is sterile normal saline solution. Each cubic centimetre contains one-fiftieth grain of parathyroid nucleoproteid and a trace of chloroform (antiseptic). This method of solution mixes English and metric systems, but practically it is very convenient. Kept in the ice-box, the nucleo-solution retains its efficiency for at least six weeks or two months, and probably much longer.

OTHER REMEDIAL RESOURCES

If the patient has gotten any relief from hyoscine before beginning the parathyroid treatment, he should continue it afterward. The two work well enough together. The dose of hyoscine, its successes and its failures, have been written up many times before. I need only comment here upon the necessity of using a pure drug. The dose of hyoscine in paralysis agitans is often made needlessly large. One two-hundredth grain is generally effective. Occasionally duboisine may work better than hyoscine. Hyoscine makes many patients so wretched in other ways that they refuse to take it. Delirium is a common symptom, and in the later stages of the disease urinary retention with overflow may occur. This should be carefully watched for.

Of the numerous remedies for chronic constipation, enemata and one or another form of the extract of Rhamnus purshiana will prove most reliable in the long run. The patient generally finds out for himself what suits him best. Calcium salts by the mouth have produced no benefit in my hands. Hypodermically I have gotten no results except local induration and threatening abscess. Intravenously I have found no patients willing to try it. Morphine, administered hypodermically, may be given in small doses and infrequently for a long while without great harm, and its employment should not be too long postponed when other remedies fail.

Of *mechanical remedies,* passive motions, warm baths, and general massage of the limbs and trunk (*secundum artem*) are helpful, but the front of the neck should on no account be massaged; an excess of thyroid secretion is thus liberated in the system and the patient grows rapidly worse.

Active exercise is of value in all patients who can take it without too great pain or fatigue. *No exercise that over-fatigues is good.* A daily airing in a wheel chair, or a carriage (not a motor-car) is essential for those who cannot walk. For patients with sufficient means extensive travelling by easy stages is often of great benefit. The mental surroundings of the patient are very important. He should be cared for with sympathy and cheerfulness, and, in the common event of psychasthenic "fear," "nervousness," or mental depression, an appeal to the reason, a resort to the consolations of religious faith, and such other rational resources as may be available, will prove of marked assistance.

As regards diet, I have made a multitude of experiments. I have kept patients upon an exclusive meat diet, an exclusive vegetable diet, an exclusive milk diet, a diet free from calcium, a diet rich in calcium—all without the least effect upon the patient's condition. This is comparable to the curious fact that the *tetania parathyreopriva* of cabbage-fed rabbits is just as violent as that of meat-fed dogs. I have finally concluded that the patient should be encouraged to eat freely of a mixed diet—taking in moderation anything that agrees with him and does not aggravate his constipation. When stimulants have been required, aërated Scotch whiskey has given my patients more general satisfaction than aught besides.

After resort has been had patiently to a sufficiently long course of parathyroid treatment, and when this medication has been rationally supplemented by one or more of the other expedients above suggested, the patient generally attains, within six months or a year, to a stationary condition—not, I think, of "cure," but of very fair comfort—and remains thus for an indefinite period. If one recalls the pessimism with which the treatment of this dreadful disease has been universally regarded in former years, it is not too much to say that medication with properly identified parathyroid has wrought a revolution.

REFERENCES

[1] Horsley, V.: *British Medical Journal*, 1885.
[2] Lundborg: *Deutsche Zeitschrift f. Nervenheilkunde*, No. 27, 1904.
[3] Berkeley: *Medical News*, Dec. 2, 1905.

[4] Roussy, G., and Clunet, J.: *Archives de médecine expérimentales*, etc., No. 3, Mai, 1910.
[5] Jeandelise, P.: *Thèse de Nancy*, Paris, 1903.
[6] Pool, E. H.: *Annals of Surgery*, 1907.
[7] Erdheim, J.: *Mittheilungen aus dem Grenzgebiet*, etc., xvi, 1906.
[8] Beebe and Berkeley: *Journal of Medical Research*, xx, 1909.
[9] Ochsner and Thompson: "A Treatise on the Surgery and Pathology of the Thyroid and Parathyroid Glands," St. Louis, 1910.
[10] MacCallum, W. G.: *Johns Hopkins Hospital Reports*, 1908.
[11] Gley, E.: *British Medical Journal*, 1901.
[12] Vassale: *Reference in Quadri, Gazetta medica italiana*, lvii, Nos. 61 and 71, 1906.
[13] MacCallum, W. G.: *Medical News*, 1903.
[14] MacCallum, W. G.: *Centralblatt f. allegemeine Pathologie u. path. Anat.*, May, 1905.
[15] Pineles, F.: *Mittheilungen aus dem Grenzgebiet*, etc., xiv, 120, 1904–5.
[16] Camp, C. D.: *Journal of the American Medical Association*, April 13, 1907.
[17] Thompson, R. L.: *Journal of Medical Research*, 1906.
[18] Berkeley: *Presbyterian Hospital Reports*, 1906.
[19] Alquier: *Gazette des Hôpitaux*, 1909.
[20] Morel, L.: *Les Parathyroïdes*, Paris, 1912.
[21] Berkeley: *New York Medical Journal*, Nov. 23, 1907.

THE TREATMENT OF DIABETES*

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In the treatment of chronic diseases it may safely be said that we frequently fail to obtain the best results, not so much from the limitations of our art to meet the problems presented as from difficulties surrounding the individual case, at times unavoidable but often self-imposed. Among these it may be the difficulty of impressing the patient sufficiently with the seriousness of his condition, to have him submit himself to the necessary régime, with its attendant expense, loss of time, and possibly long-continued self-discipline, without at the same time unduly alarming him, and so producing an unhappiness and morbid introspection which are sure to react unfavorably on the disease; or it may be the difficulty of obtaining the best opportunities for working out the details of a case, or of having the necessary supervision for its future management. It is becoming more and more recognized that in chronic diseases a casual consultation, a hurried diagnosis, a few verbal instructions, and a prescription do not fairly represent the limit of possibilities to obtain the best results, and yet these are precisely the means by which patients often expect their physician to work a cure. What has been done in the case of tuberculosis should be undertaken in other chronic diseases, viz., to educate the public to see the necessity for not only ample advice and instruction of the patient, but for such thorough training as will enable him to intelligently carry out the methods advised. The physician on his part must have not only the necessary technical knowledge and experience, but he must be ready to give time and personal attention to details even though the pressure of daily routine may make this difficult. In contrast to the surgeon, from whom we may learn a valuable lesson in this regard, physicians often fail to insist on the same favorable opportunities for applying their methods regard-

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less of the trouble and expense involved, and do not exact a similar rigidity of technique in the details of management.

In probably no disease are the difficulties and shortcomings before mentioned more apparent than in the treatment of diabetes. The too common practice, once this disease has been diagnosed, of instituting a routine plan for treatment in which carbohydrate foods are eliminated from the diet as far as possible, and then prescribing opium or other drugs, with little regard to the continued close observation of the case, to determine the patient's response to the treatment, cannot be too strongly deprecated. While such haphazard means may at times be of value, they most frequently fail, and may do more harm than good by exposing the patient to serious, though often avoidable, dangers.

While accepting the general proposition that a knowledge of the etiology and pathogenesis of a disease affords the only foundation upon which to erect a scientific plan of treatment, yet even in diseases such as diabetes, where these ultimate factors are as yet unknown, experimental and clinical investigations have succeeded in establishing many facts with regard to the course of the disease, the influence of environment, of diet, and other measures which, in the meantime, serve as valuable guides to its management. We may even recognize at times that therapeutic despair is born of too close attention to the anatomical basis of disease, which, viewing the final ruin, often tends to blind one to the many compensatory mechanisms possessed by the living tissues, and which tends to make the morbid anatomist a gloomy prognostician, and consequently unpossessed of the mental attitude to inspire the hope and confidence requisite for successful treatment.

While as yet the morbid processes underlying diabetes mellitus are not fully known, and though the prognosis is no doubt to a large extent determined by the nature and degree of these changes, yet in practice we may recognize the disease as a disturbance of metabolism, manifesting itself in a reduced capacity to assimilate carbohydrates, associated with hyperglycæmia and the appearance of glucose in the urine. Without knowing the ultimate causes of this functional incapacity, we are warranted, as a general proposition, in considering the *degree in which it manifests itself* (as shown by the amount of sugar excreted in the urine on a measured diet), and

the attendant nutritional disturbances, as an index to the seriousness of the underlying pathological condition. Considering the disease, then, from the functional side, we institute a plan of treatment which will spare the embarrassed function as far as is consistent with the needs of the organism as a whole, for reasons similar to those which induce us to reduce the strain on a weakened myocardium, or to save the inflamed kidneys by a suitable dietary. This is in accordance with Hoffman's law that "by lessening the work of any diseased function the latter may be improved." When we have succeeded, by lessening the functional strain, in causing a disappearance of symptoms, we have not only given a better opportunity for the intervention of those compensatory processes which make for readjustment and repair, but we avoid the train of *secondary* changes following in the wake of the disease. In diabetes these secondary changes due to the excess of sugar in the blood, and its excretion in the urine, are of such far-reaching importance as often to overshadow the primary disease. One need only mention the nutritional loss in the excreted sugar, amounting at times to over half the caloric value of the diet; to the reduced resistance to infections, to the occurrence of pruritis, cataract, neuritis, nephritis, cardiovascular disease, lipæmia, and, finally, acidosis and resulting coma, all of which are traceable to the hyperglycæmia.

The diagnosis of diabetes involves (*a*) the demonstration of glucose in the urine, (*b*) that it is not due merely to a temporary *excess of sugar in the diet, i.e.,* alimentary glycosuria, (*c*) the determination of the patient's carbohydrate tolerance, (*d*) the demonstration of the presence or absence of acidosis and its degree.

In testing for sugar with the ordinary Fehling's solution or its modifications, one must never forget that the reaction is that for an active reducing agent, and not for sugar *per se,* and that urine frequently contains other substances capable of reducing the copper salts, which may cause confusion or doubt. Among these substances may be mentioned excess of urates or the presence of maltose, lactose, pentose, alkaptone, glycuronic acid, etc.

From my experience in life insurance work, I find that difficulties from this source are of frequent occurrence. In every doubtful case, therefore, the common fermentation test should be applied.

One must also determine whether the case be one of alimentary

glycosuria or a true diabetes, and for this purpose all that is necessary is to put the patient on a *sugar-free diet* and then test the urine. If sugar is present on this diet, the case is one of true diabetes, as an alimentary glycosuria never occurs when the carbohydrates are given in the form of starch, as the slowness of absorption of the latter prevents a large and sudden access of sugar to the portal blood which otherwise might overtax the warehousing capacity of the liver.

In a suspected case of diabetes we may further readily test the patient's capacity to metabolize carbohydrates by the administration of 100 grains of glucose in coffee on an empty stomach and examination of the urine after two hours and then every six hours for the next twenty-four hours. If a reaction for sugar is obtained after the first two hours, it points to a reduced functional capacity to metabolize this form of carbohydrate, for it has been established by abundant clinical investigation that normal individuals can assimilate from 100 to 250 grammes of grape-sugar taken in this way. This simple test will enable one to detect cases of diabetes *in posse,* or cases (as sometimes happens in applicants for life insurance) where the sugar has disappeared from the urine owing to temporary restriction of diet.

It must never be forgotten that the labelling of a case as diabetes is a very incomplete diagnosis, and in every instance it is equally important to determine the *degree of functional incapacity* that exists. For clinical purposes cases may be usefully classified as mild, moderately severe, severe, and very severe, in accordance with their response to test diets. For this purpose the patient may be given a fat-protein diet with the addition of 60 grammes of carbohydrate (4 ounces of ordinary white bread). In Von Noorden's clinic the patient is put on the following test diet for three days: 250 grammes of meat, 150 grammes of butter, 4 eggs, 300 grammes vegetables (green); bouillon, tea or coffee or light wine, and 75 grammes of white bread. If the urine becomes sugar-free on this diet after two or three days the case is classed as mild. If the urine still contains sugar but less in quantity than the amount of carbohydrate ingested—say 30 grammes *per diem*—it shows a positive carbohydrate balance, or, in other words, some capacity on the part of the patient to assimilate this form of food. The bread may then

be reduced one-half or entirely eliminated, and if the sugar entirely disappears after two or three days on a strict fat-protein diet, the case would be classed as moderately severe; if sugar still remains, the case is a severe one.

It is scarcely necessary here to caution against the too sudden elimination of carbohydrates from the diet of emaciated patients or those showing signs of marked acidosis, on account of the danger of precipitating diabetic coma. The value of the full use of alkalies at this period will be referred to later.

In every case presenting himself for advice a complete history should be taken, a thorough physical examination made, a detail of the diet which he is taking submitted by the patient, and a qualitative and quantitative examination of a 24-hour specimen of the urine made. The clinical classification before mentioned may be modified in accordance with the data obtained in this way, nor must we forget the prognostic significance of the age at which the disease first manifests itself. Generally speaking, the younger the patient, the worse the outlook. Naunyn, Falta, Joslin, and others report having seen cures in children, though such is very unusual.

Before undertaking the treatment of a case one should have a clear understanding of the principles underlying its management and the objects to be aimed at. It is now generally recognized that the dietary is the all-important means of control, and that drugs are of very little value. As first demonstrated by Naunyn and since abundantly confirmed by others, *if the urine can be rendered sugar-free and kept sugar-free,* on a suitable dietary, without incurring unwarranted dangers, it is usually possible to gradually build up the patient's carbohydrate tolerance. This is the key to the treatment of diabetes. On the other hand, so long as the urine contains sugar the tendency is for the amount to gradually increase, even though the same dietetic restriction is maintained. We must, therefore, if at all possible, *get the urine sugar-free and keep it so.* Besides keeping the urine sugar-free, however, the diet should fulfil the following indications:

(1) Maintain the patient's weight and general nutrition (except for a short period at the beginning of treatment, when undernutrition is usually unavoidable).

(2) Preserve his strength and well-being and avoid digestive disturbance, diarrhœa, etc.

(3) Supply sufficient oxidizable material to avoid dangerous acidosis.

As the reduced capacity to warehouse carbohydrates is usually a permanent one—Naunyn thinks of hereditary origin—even in the mildest cases this function must be spared unnecessary strain for the rest of the patient's life. The prime necessity, therefore, of so thoroughly instructing and training the patient that he may be guided as safely as possible between the dangers of an excess on the one hand, which may cause a return or an increase of the glycosuria, and a restriction on the other hand, which will lead to undernutrition and the development of a dangerous acidosis, is a problem at the best requiring the most careful individual consideration and the nicest discrimination, but, unfortunately, too often without a satisfactory solution. When one considers the seriousness of the disease, the usual permanency of the reduced functional capacity, the necessity for individualizing, and the importance as well as the difficulty of the problems to be worked out, the necessity for a few weeks' or a month's close observation at the commencement of treatment, preferably in a suitable institution, or at least with a competent nurse, becomes readily apparent.

The control of the case is greatly simplified if weights and measures are recorded by the metric system, as percentages are readily calculated and food-values easily worked out. I have found no difficulty in instructing any intelligent nurse to do this. A chart should be kept in which daily summaries of the diet, the detailed results of urinalyses, the medical treatment, the patient's weight, etc., are recorded in parallel columns, so that the relations between diet and its results on the urinary excretion and general nutrition are continually in evidence. During the preliminary observation the patient is taught how to weigh his food, and it is remarkable how soon he learns in this way to estimate quantities, so that after a time he becomes sufficiently accurate that the weighing may be largely dispensed with. When the patient has returned to his ordinary duties, examination of the urine should be made at intervals varying with the severity of the case, and in every instance a detailed diet list of the day preceding the analysis should be sub-

mitted with the specimen of urine, so as to correlate the food with the urinalysis.

In mild cases the treatment presents little difficulty, a moderate reduction of carbohydrate being often all that is necessary. It is important, however, not to restrict the diet unnecessarily beyond the point where the patient is continually kept sugar-free. The patient's carbohydrate tolerance is tested from time to time, and he may be given, say, two-thirds of the amount of carbohydrate he can assimilate, the form being varied so that the diet may not become too monotonous.

In a moderately severe case, *i.e.,* one that becomes sugar-free after a few days' fat-protein diet, if the weight and general strength can be maintained and symptoms of acidosis avoided, he should be kept for some weeks on this strict diet and then a small amount of carbohydrate added and the tolerance tested by urinary examination. A small amount of cream (4 ounces), a half grape-fruit or orange, and, finally, 30 grammes of bread, then 60 and 90 grammes, and, finally, substitutes for bread in an equal amount of other carbohydrate, as oatmeal, potato, etc., should be tried. The gradual working out of a satisfactory diet requires time and patience.

In the severe cases—that is, those that do not become sugar-free on a fat-protein diet—a much more difficult problem is presented. In these cases the patient elaborates sugar from the carbohydrate portion of the protein molecule so that in some cases as much as 80 per cent. of the ingested proteid is changed into sugar and even the body proteids may be attacked. Such patients at times may show an intolerance for proteid equal to that of the carbohydrate (Naunyn), so that a *restriction of proteid food in these cases becomes an essential part of the treatment.* The range of dietary, therefore, becomes greatly restricted, and the closest watch must be kept for signs of dangerous acidosis, and, though we should persist to the limit of prudence to render the urine sugar-free, this must not be done at the danger of producing diabetic coma or too marked under-nutrition. The greatest judgment must be exercised in these cases, but with care and patience one may often have the satisfaction of seeing a severe case assume a mild or moderately severe form. To render these cases sugar-free, starvation days, fat-vegetable days, or at times oatmeal days, or a series of these in

conjunction may be resorted to. Time will not permit of a full discussion of the application and value of these methods, a trial of which will convince one of their usefulness in properly-selected cases. In very severe cases where all attempts to render the urine sugar-free or establish any degree of tolerance have failed, the persistence in heroic dietetic methods is not warranted. It is better to allow a moderate amount of carbohydrate and proteid and be guided more by the patient's general nutrition and the acidosis than by the glycosuria. An occasional starvation day, vegetable day, or oatmeal day may help to hold back the increasing glycosuria, as well as to reduce the acidosis.

The consensus of opinion of those who have experience with these special diets is that they mark the most important advancement of recent years in the management of the disease. Under-nutrition for short periods is unavoidable in the treatment of severe cases, but this must not be carried too far. During the period of under-nutrition, especially on vegetable and starvation days, or during prolonged strict fat-protein diet, alkalies should be freely administered. For this purpose bicarbonate of soda in Vichy water or soda water, up to an ounce or more daily, or until the urine has been rendered slightly alkaline, should be taken, if the patient can tolerate it. Von Noorden recommends calcium chloride as well, on account of its action in binding intestinal acids which would otherwise be absorbed. Saline aperients, as Carlsbad salts, to keep the bowels active, are of value, but should not be used so freely as to produce local irritation or diarrhœa. Such symptoms as air hunger, digestive disturbance, diarrhœa, flatulency, vomiting, insomnia, restlessness, sleepiness, or rapidity or irregularity of the pulse are danger signals which should never be ignored. Daily tests for acetone, or, what is even easier, the ferric-chloride test for diacetic acid, should be made. This test is so simple that the nurse, or even a member of the family, if necessary, can be readily instructed to carry it out. While of value in indicating the presence of diacetic acid, its significance must not be overestimated, as the reaction may be for months or years without any dangerous symptoms supervening. One must not forget that where salicylates are being administered the urine gives the ferric-chloride reaction, which, however, does not disappear on boiling the urine, as it does with diacetic acid.

The treatment of the extreme cases of acid intoxication ending in coma is very unsatisfactory. Alkalies are our mainstay and should be used as freely as possible. With symptoms of impending coma, rest, diet, etc., withdrawal of blood from a vein and the injection of an equal amount of 4 per cent. bicarbonate of soda solution (1 litre), with or without the use of a similar amount of a solution of lævulose, is the plan followed in the Noorden clinic, and temporarily the results are remarkable. Falta has reported a patient alive three months after his restoration from coma by these means. All authorities are agreed that it is much easier to prevent acidosis than to get rid of it once it has developed. As in cases of inanition, the condition is due to the noncombustion of carbohydrates, the unneutralized acid products of incomplete metabolism accumulating in the system. In health and in mild cases of diabetes the reserve glycogen in the liver supplies a sufficient amount of readily oxidizable material to insure combustion. The margin of safety at any time is closely related to the glycogen reserve, which has been shown experimentally to be decreased in proportion to the degree of glycosuria, the restriction of diet, the amount of emaciation of the patient, etc. In cases of impending coma, therefore, the patient should be given milk, gruel, and other readily oxidizable carbohydrates freely. Allard has shown that a "hunger day" may reduce the acidosis greatly, and this should be followed by a diet low in both fats and proteins. Strauss, Blum, and other German authors speak highly of inulin as an available carbohydrate, especially in severe cases.

Apart from their value in symptomatic treatment, drugs have a very limited use in the therapy of diabetes. Those with the best reputation, such as opium, belladonna, antipyrine, etc., have their effect through a sedative influence exerted upon the nervous system, or by retarding absorption of carbohydrates from the intestinal tract (opium), but they do not increase the patient's tolerance for carbohydrates. They are of most value in neurasthenic diabetics (Von Noorden). I have never seen any benefit from yeast, pancreatic extracts, or other substance given with a view to acting upon the digestion of carbohydrates in the duodenum. Organotherapy, from which much was hoped, has been disappointing.

The importance of avoiding worry, physical and nervous strain or fatigue, cannot be too strongly insisted upon. An apprehensive, introspective patient presents a difficult clinical problem, and in very severe cases, which cannot be controlled by diet, it is often better to leave the patient alone than to worry him by fruitless attempts to get rid of the sugar in the urine. I have recently had a case where a patient was sugar-free, and, without alteration in his diet, developed a glycosuria of 8 per cent., owing to acute business worry. Pleasant occupation and moderate exercise are of advantage in maintaining the general nutrition and furthering oxidation. It is scarcely necessary to speak of the seriousness of even trifling operations in chronic diabetes, often tending to precipitate an attack of coma. The same applies to attacks of vomiting, diarrhœa, intercurrent diseases to which the patient is subject owing to his reduced capacity to resist bacterial infection.

The danger of too copious ingestion of water—especially carbonated—in producing sudden gastric or cardiac dilatation should be kept in mind.

In mild cases a visit to Carlsbad, Neunahr, or Vichy, with their pleasant surroundings and opportunities for appropriate dieting, combined with the alkaline waters, is often of much value, but severe cases should never undertake the uncertainties of diet, fatigue, and worry incident to prolonged journeys.

The results of treatment are naturally best in the mild case and in those that come under treatment early before advanced secondary changes have developed.

The occasional association of diabetes with Graves's disease has suggested a relationship between the ductless glands and some cases of diabetes. This has been studied by Falta and others, who have thus brought forward many facts in favor of this idea.

May not the effect of the subcutaneous transplantation of portions of pancreas in experimental pancreatic diabetes dimly foreshadow a possibility, made approachable by Carrel's success in the cultivation of living tissues in artificial media and his successful transplantation of organs, that a future plan of attack may be based on these lines?

REFERENCES

[1] Von Noorden: "Die Zuckerkrankheit u. ihre Behandlung," Berlin, 1910.
[2] Naunyn: "Diabetes Mellitus," *Modern Clin. Med.*, vol. i, p. 75, 1910.
[3] Falta: *The Harvey Lectures*, 1908–9, p. 97.
[4] Graham Lusk: "Metabolism in Diabetes," *Harvey Lectures*, 1908–9.
[5] Elliott P. Joslin: "Diabetes Mellitus: Hare's Modern Treatment," vol. ii, p. 427 *et seq.*, 1911.
[6] Theodore C. Janeway: "Diabetes Mellitus: Practical Treatment," vol. ii, p. 1, 1912.
[7] L. Blum: "Neuere Ergeb. d. Therap. d. Diabetes Mellitus," *Therap. Monatshefte*, Heft 4, 1912.
[8] H. Strauss: "Ueber Kohlenhydratkuren bei Diabetikern," *Klin. Vortrag Deut. med. Woch.*, den 7 Marz, 1912.
[9] Henry Sewall: *Amer. Jour. Med. Sci.*, cxlii, No. 3, 1911.
[10] W. Falta: "Die Therapie des Diabetes Mellitus," *Ergeb. d. inn. Med.*, 1908, vol. xi.
[11] Alexis Carrel: "On the Permanent Life of Tissues Outside of the Organism," *Jour. Exper. Med.*, vol. xv, No. 5.

CLINICAL INFERENCES TO BE DRAWN FROM THE WASSERMANN TEST

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The Wassermann serum test is a ferment reaction, whose rational technical basis rests on the laws governing ferment activity. The different substances entering into a ferment reaction, even if they could be prepared in an absolutely pure state, and even if the reaction itself were absolutely specific, do not bear any striking quantitative relationship to each other, because the activity of a ferment under given conditions depends principally on its concentration, and to a limited degree only on the absolute amount that is present. For instance, other conditions being constant, the same amount of complement that will dissolve several units of sensitized blood-corpuscles will dissolve only one, if the amount of physiological salt solution is increased accordingly. Furthermore, an increase in the "amboceptors" permits the use of a smaller amount of complement to do the same work in the same length of time.

THE COMPLEMENT

The *complement* is a ferment contained in the blood-serum of most, if not all, animals. We have not learned as yet to separate it from the serum. The strength of this ferment varies within wide limits from week to week and even from day to day, according to the general health of the animal. Thus, I have found that syphilitics at the height of their infection have only one-quarter to one-half as much native complement as has the average healthy individual.

If complement is added to a mixture of antigen and antibody, it uses itself up in the process of destroying the sensitized antigen, but it is not bound nor does it constitute an essential part of the products of this reaction. The manner in which the complement dis-

appears is somewhat analogous with the disappearance of the ferment pepsin from an acidulated protein mixture. The pepsin spends its chemical energy in the process of converting protein substances into albuminoses, but it does not as such take part in the formation of these substances. The amount of work that a certain quantity of pepsin does depends, other conditions being the same, principally on its concentration.

The action of the complement on the "sensitized syphilitic antigen" does not produce any unmistakable and easily appreciable optical changes. But, as the complement will be absent from the mixture if a reaction has taken place, we can determine the presence or absence of this ferment in an indirect way; that is, by the addition of a sensitized antigen whose destruction produces gross optical changes. Such an antigen is the red corpuscle of a warm-blooded animal. Dr. Wassermann was the first to make use of the complement deviation in the diagnosis of syphilis, and of its indirect determination by using the red corpuscles as an indicator. He uses the red blood-corpuscles of the sheep, but the blood-corpuscles of almost any animal species can be used, provided their peculiarities in relation to the human serum are known to the person who makes the test. It is of importance, however, that the animal from which the blood-corpuscles are taken for the reaction should be healthy and should, above all, not be suffering from any chronic affection, such as chronic malaria, chronic alcoholism, tuberculosis, or syphilis, because some animals with these chronic affections render the blood-corpuscles more resistant against the complement action.

THE ANTIGEN

What we call "antigen" in the Wassermann test, and what we extract from non-syphilitic as well as syphilitic livers, is, in all probability, a normally present non-specific protecting substance of the animal organism. The extracts of animals which are not subject to syphilitic infection can also be used in the diagnosis of syphilis. Experience has shown that they do not react with non-syphilitic sera, and it has also been shown that the watery extract of spirochactæ, of syphilis and recurrent fever, as well as other bacteria, have "antigenic" value. It is, therefore, possible, if not probable, that we are dealing with a number of reactions in the same test.

At all events, it is a wise precaution, until we know something more definite about the chemistry and biology of the test, to use syphilitic livers only to make the antigen.

THE HUMAN SERUM

It is not the purpose of this paper to discuss the question whether the human system forms a specific antibody in response to the syphilitic invasion or if the blood-serum of syphilitics contains simply an excess of waste material (toxin) and "lipoid substances" with a minus of complement and "antisheep amboceptor." The fact remains—and it has been proved by hundreds of thousands of tests—that, in the presence of syphilitic liver extract, the blood-serum of a patient suffering to any extensive degree from active syphilis will cause a deviation of the complement in larger quantities than the blood-serum of patients suffering from any other affection. Wassermann thought that the relation of the different substances entering this test was quantitative. He did not look upon the complement as a ferment that acts according to its concentration, and that rapidly loses its power to act if diluted. This serum reaction, therefore, can be made more sensitive and more accurate if we use the different ingredients in a more concentrated form than has been advised by its originator. The different substances entering this reaction should be used in such a concentrated mixture as is compatible and feasible with accurate measurement. An excess of physiological salt solution can be added after the reaction has taken place.

While studying the nature of the positive Wassermann reaction in the first stages of syphilis, and while investigating the extent to which changed outside conditions, a change in the surroundings of the patient influenced the degree of the Wassermann reaction, it was necessary to observe and compare small variations in the complement fixation. I used, therefore, the above-indicated modification. The test is made according to Wassermann's original method, but the amount of physiological salt solution mixed with the substances entering this reaction is limited for standardizing to the absolutely necessary quantity. The moment the negative control is completely hemolyzed, which should take place within one to two hours, further action on the part of the complement is stopped in all the tubes at

once by the addition of an excess of physiological salt solution. With the aid of this slight modification of the technic of the test, I have examined syphilitics, who were not under treatment, at intervals of three days to a week, and found that the reaction changes uniformly to a slight degree according to the variation in the sanitary and hygienic surroundings. Outdoor exercise and good habits will decrease the extent of a positive reaction, while indoor work and bad habits—smoking and drinking—will increase the degree of the reaction.

In chancres I found that the reaction increases gradually, and that already, when the initial lesion first appears, there is some inhibition to hæmolysis. Furthermore, it was found that persons suffering from syphilis have less than the average amount of native complement and natural antisheep "amboceptor." A syphilitic at the height of the infection shows seldom more than 25 per cent. of the average amount of complement. The natural antisheep "amboceptor" decreases in about the same ratio as the complement, but the changes are not brought about quite as rapidly as with the latter. Persons suffering from chronic affections other than syphilis show usually also less than the average amount of complement and antisheep "amboceptor." Even in perfectly healthy individuals the Wassermann reaction is subject to changes, depending solely on outside conditions. For instance, in a medical student, whose blood I used from time to time as a negative control, the blood changed to faintly positive after he had entered a hospital. A young woman of delicate and anæmic type changed from a weakly positive to a negative reaction after her hygienic and dietetic conditions were improved.

There is no doubt about the great clinical value of the Wassermann test, but it has its limitations; a negative reaction may be observed in all stages of syphilis and a partial or a distinctly but not strongly positive reaction may be observed in many non-specific, usually chronic, affections. Some of those affections are: malaria, carcinoma, sarcoma, recurrent fever, and narcose. Furthermore, a strongly positive Wassermann reaction does not exclude carcinoma or sarcoma, since it is well known that syphilis is apt to prepare the soil for such growth, especially for the former (carcinoma).

In the primary stage of syphilis the reaction remains negative for from one to three weeks after the appearance of the initial lesion.

The reaction appears gradually; the increase can be observed by examining the blood-serum every three to four days, beginning with the first appearance of the primary sore. The diagnosis usually can be made at the time of the third examination; that is, a week to eight days after the appearance of the initial sore.

THE EFFECT OF SALVARSAN ON THE WASSERMANN REACTION

An injection of salvarsan, and to a lesser degree big doses of mercury, given to a patient with an initial lesion and a negative Wassermann reaction will shorten the time otherwise necessary for the production of a positive reaction. This is caused by the injurious effects of our therapeutic aids upon the host. Large doses of anti-syphilitic drugs given at this stage of the disease apparently depress the vital resistance of the patient to a greater extent than they injure the disease-producing micro-organism. The latter multiplies more rapidly and the invasion of the human body as a whole is accomplished within shorter time than without treatment. But it is, too, this increased and injurious depression which arouses the human body to make an increased effort at the defence, and it is in this way, in part at least, responsible for the ultimate production of a negative Wassermann reaction. The negative reaction is the result of the combined injury of drug and disease, supported by the vital resistance of the human body. Professor Ehrlich gave us in salvarsan an arsenic compound which shows practically no dissociation of arsenic in saline solution outside of the animal body and which is very slowly and in different individuals with varying degree of quickness broken down by the human system. It is not an organic and specific compound in the true sense of the word, such as tuberculin is for tuberculosis. The therapeutic value of "606" depends solely upon its contents of arsenic. Now, we fully admit that it is the mildness of the tuberculous infection, the mildness of leprosy, the mildness of the syphilitic infection which allows these affections to become chronic and which makes them ultimately so dangerous to the life of the patient.

We also admit that the human system can be stimulated to make a defence by injury only, and we further admit that the defence of the diseased human system will, other conditions being the same, be the more vigorous, the more acute and severe the injury is; but we do not believe that any unbiased, broadly-trained scientific person

could ever have entertained a serious thought to sterilize, to free completely the human body of syphilis, or of any other disease to which mankind is easily susceptible, by a dose of an arsenic or any other compound, without at the same time permanently harming or even destroying the patient.

The beneficial effect of an injection of salvarsan is clinically very apparent, while its detrimental effect, being an insidious fatty degeneration, is not at all conspicuous, but nevertheless it is full of grave consequences. Mercury is a far safer, a far less dangerous, and a far more efficacious antisyphilitic drug than any arsenic compound made or to be made, for the simple reason that the detrimental effect of mercury on the human system becomes very apparent and acute long before it has a chance to cause really serious damage to the patient.

Ehrlich's drug experiments are correct and scientific by themselves, but they are for various reasons unscientific and misleading in their application to the human system.

Dr. G. Frank Lydston, a syphilographer of very large experience, and a man whose training rests on a broader basis and whose conclusions embrace a larger mental horizon than we would expect of the average specialist in this line of work, says: "There will be more uncured syphilis at the end of the next decade, and more suffering from the sequelæ due to the combined effect of syphilis and its treatment, than the routine users of salvarsan dream of; this aside from the disastrous results due to overconfidence in the new drug."

EFFECT OF TREATMENT IN THE WASSERMANN TEST

In the secondary and tertiary cases of syphilis, which were until recently, or are at the present time, under antisyphilitic treatment, the Wassermann reaction may be negative and discredit the clinical diagnosis. There is no doubt that many times in the past wrong has been done in this way to the practical clinician. The positive Wassermann reaction disappears under treatment quicker in the early than in the later stages of the disease, but it is also more likely to return after a short interval of time. A negative Wassermann in syphilitics who have been treated can sometimes be made positive again by an injection of salvarsan. The factors which cause the return of the reaction are identical with the factors that cause an

early appearance of the positive reaction in the primary stage. If the reaction returns to positive it means that the patient still has syphilis, but syphilis cannot be excluded even if the reaction remains negative. The patient may still be infected, but either the virulence and the vegetative properties of the spirochæta have been reduced or the host has gained in vital resistance.

SYPHILIS, WITH A NEGATIVE WASSERMANN REACTION

In syphilitic patients who have been rendered negative with the aid of mercury and potassium iodide the reappearance of the Wassermann reaction precedes the return of clinical symptoms, while in the patients treated with large doses of arsenic, no matter in what form (salvarsan, etc.), the clinical symptoms not infrequently reappear while the patients still show a negative Wassermann. This phenomenon stands probably in some relation to the fatty degeneration produced by the administration of large doses of arsenic compounds.

In acquired syphilis, which has not been treated at all, a negative Wassermann reaction is occasionally met, but very rarely. In congenital syphilis with infection of the male parent only, the negative reaction amounts to about twenty per cent. of the clinically luetic infants.

EXAMINATION OF SPINAL FLUID

In suspected syphilis of the nervous system the spinal fluid should be examined if the blood-serum is found negative. While a positive Wassermann of the blood-serum means luetic infection, in a general way a positive spinal fluid reaction means a local condition; namely, the diagnosis of a luetic disease of the central nervous system. In these cases the spinal fluid alone may show a positive reaction, the blood-serum being negative.

A positive Wassermann reaction of the spinal fluid proves luetic infection and explains a paraluetic condition, such as general paralysis, tabes, etc. The lumbar fluid does not contain any complement and needs not to be inactivated, and it should be examined in increasing doses, using 1, 2, 3, and 4 units.

CONCLUDING REMARKS

It is, at the present time, more or less generally accepted that an early and energetic treatment is the most likely to give a permanently negative Wassermann reaction, but even if we take the truth of this

statement for granted, and even if we take for granted, also, that a "permanently" negative Wassermann reaction means absence of syphilis, it must be remembered that the objects of our therapy include, besides killing the *spirochæta pallida,* the preservation of the patient. What I mean by this can be more clearly understood by citing an example. I have under observation two patients of about the same age, sex, and occupation, who contracted syphilis about fourteen to fifteen years ago. One patient took a very thorough treatment for three to four years, and I have examined his blood a number of times the last few years and always found it negative. The other patient did not tolerate medical treatment well, but he was willing, however, to follow any hygienic and sanitary measures suggested. I took special care to teach him how to live and how to raise his resistance as a protection against being used as a fertile culture media by the syphilitic virus. The blood of the latter patient is still positive to-day, fourteen years after the appearance of the initial lesion, but he enjoys otherwise perfect health. He does not show any outward signs of syphilis; he is in the best of physical condition, while my negative patient is in delicate health and physically a relatively old man.

For the last two months I have been making experiments with arterial and venous blood taken from the same patient at the same time and tested for the Wassermann reaction under exactly the same conditions and find that venous blood deviates more complement than arterial. The difference has never been large enough to essentially change the results of typical negative or positive cases, but a difference is always present and should be taken in consideration when reading the results of cases with doubtful reactions.

In concluding I would say that the correctness of the results of the Wassermann reaction grows with the number of daily tests and with the experience of the serologist making the examinations. The best guaranty for the clinician to get a clinically valuable Wassermann report is one from a centralized laboratory, where tests are continuously being made.

TREATMENT OF EXOPHTHALMIC GOITRE *

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The study of the internal secretions constitutes one of the most important epochs in revolutionary and evolutionary medicine. The glands of internal secretion not only detoxicate certain products of metabolism, but furnish hormones which stimulate anabolic and catabolic processes and furnish tone to the autonomic and sympathetic systems.

In 1859 Schiff noted fatal results in dogs after thyroidectomy, and a *cachexia strumipriva* was observed by Kocher after the same operation in humans. Gull and Ord demonstrated the relation of the thyroid gland to myxœdema, and Murray showed that the latter and cretinism yielded to thyroid feeding. Others noted the relief of symptoms in thyroidectomized animals following subcutaneous transplantation of the gland.

Symptoms, notably tetany, following thyroidectomy are due to injury or removal of the parathyroid glands (two small pairs of glands situated behind the lateral lobes of the thyroids in juxtaposition to the trachea). The parathyroids are supposed to regulate calcium metabolism.

Revivescency of the thymus gland has been noted in exophthalmic goitre, and implantation of this gland in dogs has been followed by tachycardia and exophthalmos.

In my last lecture [1] vagus tone was discussed, and reference was made to several diseases caused by vagus-hypotonia. The aphorism, "*Naturam morborum curationes ostendunt,*" justifies my theory of exophthalmic goitre, viz., that it is caused by hypotonia of the vagus.

The symptoms are supposedly caused by a hypersecretion of the thyroid gland conducing to a species of chronic intoxication. This hypothesis is justified by the enlarged gland which shows increased vascularity and secreting epithelium. Furthermore, the symptomatology of the disease may be reproduced by thyroid feeding and re-

* Address before "The American Association for Study of Spondylotherapy."

[1] "The Tonus of the Vagus," INTERNATIONAL CLINICS, vol. iii, 22d Series.

moval of the gland, and the surgical diminution of its vascularity means, as a rule, the relief of symptoms. The vascularity of the gland is enormous. Every minute the blood passing through it is equivalent to six times its weight, and it is about 28 times as vascular as the head and 5½ times as vascular as the kidney.

The thyroid provokes symptoms arising from deficiency or excess of its internal secretion, or from irregular functional activity (dysthyroidism).

The most important principle isolated from the gland is iodothyrin. The latter is characterized by its high content of iodine, combined with albumin and globulin. The iodine in the gland was first demonstrated by Baumann in 1896. Iodine is practically absent in other tissues, and its amount in the thyroid varies with the species and individual. Insomuch as most vegetables contain iodine, it is most abundant in herbivora and least in amount in carnivora. The iodine content is increased by the administration of potassium iodide, and decreased by an exclusive diet of meat. Iodoform poisoning presents symptoms suggestive of thyroid intoxication, and in animals dosed with iodoform the iodine content of the thyroid gland is augmented.

In all goitres, excepting exophthalmic goitre, the quantity of iodine in the gland is reduced. The normal thyroid of man and domestic animals contains arsenic, and thyroidism from thyroid feeding may be prevented or alleviated by the concurrent use of Fowler's solution (3 minims, three times a day).

The thyroid bears an important relation to morbid phenomena in females. It congests and enlarges with menstruation, and usually hypertrophies in pregnancy. In the latter, iodothyrin in small doses diminishes the gland.

In the pregnancy-kidney with tube casts and albuminuria thyroid hypertrophy is usually absent, and there is a tendency to eclampsia which is favorably influenced by thyroid extract. Owing to the frequency of thyroid implication in women, the gland must always be examined to explain, if possible, unusual phenomena. Hyperthyroidism is not always expressed by a typic symptomatic picture. A persistent tachycardia may be the only evidence of augmented activity of the gland. In other instances, the frontier

symptoms may be emaciation, amenorrhœa, irritability, or some mental anomaly.

Any swelling of the gland is indicated by a bulging or fulness of the neck corresponding to the situs of the gland which rises during deglutition. To facilitate exploration of the thyroid the method of Woodbury is to be adopted. This is done by examining the gland with the neck extended, and the chin being rotated nearly over to the opposite shoulder, with side of the head slightly flexed on the chest. By means of a piece of ground glass approximating the neck and head in a dark room with light at a fixed point and properly adjusted you can trace the degree of exophthalmos and struma, and the tracings can be used for future reference.

One must also search for aberrant and accessory thyroids, notably at the root of the tongue. This lingual thyroid is not uncommon. In a patient typically affected with exophthalmic goitre one notes the classic tetrad of symptoms: tachycardia, exophthalmos, tremor, and pulsating thyroid gland. But it is the recognition of minimal hyperthyroidism that taxes the diagnostic acumen of the physician. There are a number of bizarre and protean symptoms due from hyperthyroidism which parade under a medley of names and run the gamut of hysteria to psychasthenia. Many symptoms at puberty, menstruation, pregnancy, and the menopause are caused by a transitory hyperæmia of the thyroid gland. Such symptoms are accentuated by factors which increase the gland's vascularity or diminish vagus tone. Such factors are: emotional disturbances, sexual excitement, genital disturbances (chiefly uterine), infectious diseases (notably influenza), coffee, tea, alcohol, and certain drugs (iodides, iodoform, and thyroid extract).

You will understand the relation existing between hyperthyroidism and emotions by recalling my last lecture on vagus tone. One may arrive at a diagnosis of hyperthyroidism by the administration of iodine, iodothyrin, or practically any thyroid preparation. Thus, in the thyroid test, we administer ten five-grain doses of a reliable thyroid preparation. Symptoms are accentuated if caused by hyperthyroidism, and ameliorated in hypothyroidism. Antithyroid preparations would ameliorate the symptoms of hyperthyroidism, but unfortunately the specific sera are uncertain in action.

In my lecture on "vagus tone" reference was made to a diagnos-

tic test of great value in the recognition of minimal hyperthyroidism. This refers to the fact that in hyperthyroidism firm pressure for about 30 seconds at the seventh cervical spine causes little or no descent of the lower border of the lungs, whereas in the norm it descends about 4 centimetres, and in hypertonia of the vagus it may descend 6 centimetres. The instrument (Fig. 1) is applied to both sides in the region of the seventh cervical spine. In the absence of this instrument, firm pressure may be made with the thumbs. This barodiagnostic manœuvre is easily executed.

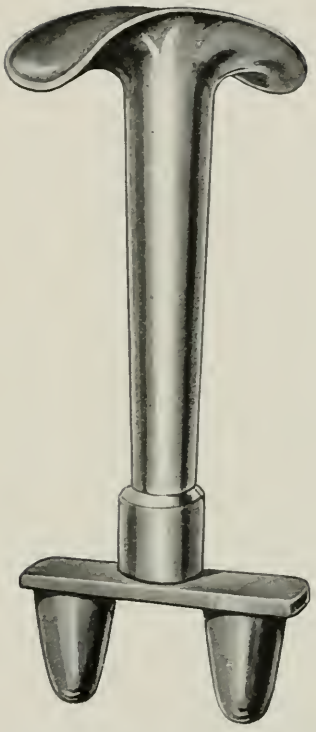
All the human emotions may be expressed through the vagus. The tone of the entire nerve may be compromised, but the brunt of increased or diminished tone may be borne by an individual branch (local vagus hypertonia or hypotonia). We can now understand why certain visceral symptoms may predominate in this disease, and, what is more, we can determine objectively with almost mathematical certainty the degree of tone of a given viscus receiving vagal innervation. You know that when pressure is executed at the seventh cervical spine the pulse may be inhibited. With this instrument, which I have neologized as spondylobaresthesiometer, I can measure the degree of pressure. It is necessary for me to establish a clinico-physiologic law, viz., the weaker the stimulus necessary to elicit cardiac inhibition the more diminished is the tone of the vagus. Thus we recognize an orthotonic, hypertonic, hypotonic, or even an atonic vagus.

In the norm, in orthotonia of the vagus, cardiac inhibition does not occur when the pressure exerted is less than 10 kilogrammes. In this patient with tachycardia the radial pulse is momentarily inhibited with a pressure of 3 kilogrammes. If I now stimulate the vagus by concussion of the seventh cervical spine, a pressure of 8 kilogrammes must be made before the pulse is temporarily inhibited.

Here is another patient with exophthalmic goitre whose predominant symptom is tachycardia. Yesterday, seven kilogrammes of pressure was necessary to stop the pulse. Last night she suffered from an intense emotion, and to-day the pulse is inhibited with a pressure of 4 kilogrammes.

I shall illustrate on a patient the immediate effects on the exophthalmos by increasing and diminishing vagus tone. (Fig.

Fig. 1.



Radicularpressor for augmenting vagus tone by pressure in the paravertebral region corresponding to the seventh cervical spine.

FIG 2.

Eyes illustrating the effects on the exophthalmos in exophthalmic goitre by increasing and diminishing vagus tone: *a*, before; *b*, during time vagus tone is diminished; and *c*, when vagus tone is increased. (Compare by looking at depression in bridge of nose caused from the wearing of eyeglasses.)

2, *a*, *b*, and *c*).[2] This test is practically diagnostic of this disease.

Firm pressure at the seventh cervical spine causes the eyes to recede, and there is often an immediate amelioration of other symptoms. The contrary condition ensues when vagus tone is depressed by pressure between the third and fourth dorsal spines.

Time will not permit me to discuss symptoms which are at your command in any text-book. I want you to comprehend some of the ocular symptoms of this disease and will ask you to study Fig. 3.

FIG. 3.

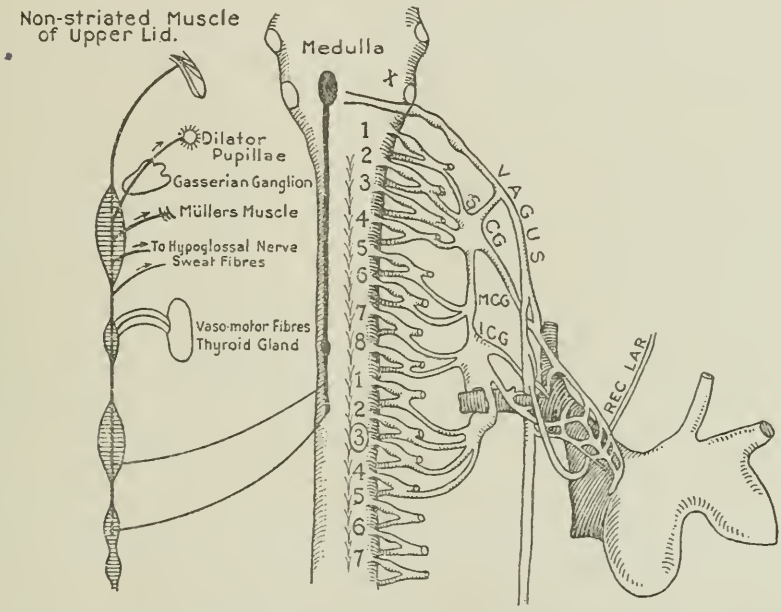


Diagram of course of oculopupillary fibres of the cervical sympathetic. The pupil-dilating fibres arise from the pupil-dilating centre in the medulla, and descending in the lateral column of the cord they emerge in the anterior roots of the first and second thoracic segments. Entering the inferior cervical ganglion by white rami communicantes, they ascend in the cervical sympathetic to the gasserian ganglion and pass to the orbit along the ophthalmic division of the trigeminus. The other half of the diagram shows the origin and course of the cardiac nerves. The stimulus applied at the seventh cervical spine corresponds to the third dorsal segment of cord.

The cervical part of the sympathetic chain containing oculopupillary fibres innervates the dilator pupillæ, Müller's muscle (attached to the bony wall of the orbit and inserted into the sclerotic coat of the eyeball and the upper or lower lids), and non-striated portion of the levator palpebræ superioris. There are also fibres to the hypoglossal nerve, and sweat and vasomotor fibres.

[2] I am indebted for these photographs to Dr. George O. Jarvis.

The ocular symptoms in this disease, viz., mydriasis, exophthalmos, widening of the palpebral aperture, and the sign of von Graefe, are caused by irritation or stimulation of the cervical sympathetic.

When I make pressure in the region to depress the vagus the sympathetic fibres become dominant in action, the result being stimulation of the non-striated fibres of the upper lid and Müller's muscle, which results in widening of the palpebral fissure, retraction of the lids, and exophthalmos. An antithetic effect (notably enophthalmos) ensues by augmenting vagus tone by pressure at the seventh cervical spine. Insomuch as the cervical sympathetic is accessible to clinical observation, it may serve as an index to the condition of the general sympathetic system and should be tested as a routine method.

By pinching the skin of the neck, the ciliospinal reflex of pupillary dilatation ensues on the same side. The cervical sympathetic may be stimulated by conjunctival instillation of a few drops of cocaine solution. As a result of this manœuvre, in our patient you will note in the right eye slight exophthalmos, mydriasis, and retraction of the upper lid.

When the reflexes of the sympathetic are increased, instillation of a drop of a one per thousand solution of adrenalin causes pronounced mydriasis. The retraction of the upper lid in this disease is caused by spasm without myasthenia.

If, to the upper lid, I attach an 8 centimetre strip of plaster to the lower end of which is fixed a 20 gramme weight, the closed lid can raise it higher than in the norm (10 to 15 centimetres), but it is raised in jerks, and this method may be utilized in noting the spasm which is expressed in the norm by retracted lids.

In doubtful cases I employ pharmacologic reactions. Ten minims of adrenalin chloride solution (1–1000) given hypodermatically will at once accentuate the exophthalmos and diminish the size of the thyroid. Pilocarpine will ameliorate both conditions. One may also have recourse in diagnosis to biochemical tests. Thyroid extract antagonizes adrenalin in its pupillodilator action on the frog's eye, and this fact may be employed in the clinical recognition of hyperthyroidism. This reaction is obtainable with the blood in exophthalmic goitre, but is negative with blood from neurasthenic and hysterical patients.

The blood in hyperthyroidism increases the resistance of mice to

poisoning with morphine and acetonitrile, thus making it possible to double the lethal dose.

The blood findings in this disease are characteristic: increase of lymphocytes and decrease of leucocytes. This blood-picture has recently been found in simple goitre.

My treatment of exophthalmic goitre, as well as for minor and atypic manifestations of hyperthryroidism, consists in increasing the tone of the vagus. In the conventional treatment, which ranges from galvanization of the cervical sympathetic and exposure to the X-rays to the use of specific sera, the results are uncertain and recurrence is the rule. Respecting thyroidectomy, the results are better, but little good is obtained by sympathectomy. My method of treatment is practically a specific, and the results are usually permanent. Recidivation is transient and usually associated with factors augmenting the vascularity of the thyroid gland. The first symptoms to yield are tachycardia, arrhythmia, nervousness, and diaphoresis. Exophthalmos is the most persistent symptom. It may yield synchronously with the other signs, it may improve after treatment is suspended, or it may be permanent. Operations give no better results. Many therapeutic methods are discredited for the reason that they are not executed in detail. In cases of long standing, exophthalmos is permanent, owing to the deposit of orbital fat, which causes the eye to protrude even though the muscle of Müller is no longer contracted.

Cases have been successfully treated by my method which have failed to yield to operative measures. The treatment[3] consists essentially of stimulation of the vagus at the seventh cervical spine. The stimulus employed is rapid concussion, although it may be substituted by the rapid sinusoidal current to the same region. An *apparatus giving a percussion blow* must be employed. You cannot elicit the knee-jerk by the conventional vibrator, hence you cannot expect it to provoke a vasoconstrictor reflex which is so essential in the treatment of this disease.

When concussion is made at the seventh cervical spine (corresponding to the third dorsal segment of cord (Fig. 3), the stimulus is conveyed primarily by the spinal nerves to the sympathetic and

[3] For the minute details of the method, the reader is referred to the author's third edition of Spondylotherapy.

vagus, which make up the superficial and deep cardiac plexus. By this method, however, the effects are secured by indirect vagus stimulation. Even after a single séance of concussion one may note a reduction in the pulse from 10 to 30 beats per minute, and likewise a diminution in the size of the struma. Within three weeks, as a rule, in recent cases (provided two séances of concussion are given daily) the symptoms of hyperthyroidism disappear.

From many reports of physicians, the results have been practically uniform. Let me read the following extract of a letter from a physician who has successfully treated many cases:

> "It is only a question of time when physicians will and must recognize your specific treatment, and when it will be regarded as criminal negligence for the physician to invoke surgery before giving your method a trial."

Among the many letters received from physicians one question is paramount: Will concussion of the seventh cervical spine cure simple forms of goitre? The reply to this question may be given as follows:

In goitres showing vascularity of the gland (soft and tender with systolic blowing or pulsation) there is some chance of reduction, but when there is fibrosis of the gland no results can be expected. Enlargement of the thyroid is frequently a compensatory phenomenon like kidney hypertrophy to compensate for the secretory deficiency of its fellow, or when polycythæmia occurs in high altitudes to make the most of the deficient supply of oxygen. Similarly, the thyroid enlarges because it is required to supply itself with iodine conveyed to it by the blood. Here iodides or thyroid extract if given early prove curative. If you study the many drugs which have been exploited for the cure of exophthalmic goitre, you may comprehend facts heretofore inexplicable.

I have neologized "clinical pharmacology" to indicate the study of pharmacology of the human in lieu of laboratory animals. My investigations show that a large number of drugs owe their physiologic and toxic action to either an increase or decrease of vagus tone.

Thus, quinine hydrobromide has been demonstrated to be of great value in this disease, but it must be used persistently for months or years. After ingestion of this drug, one may note that the vagus tone is much increased.

In my opinion, *pilocarpine* is the most efficient drug in this and other affections caused by hypotonia of the vagus. Given either hypodermatically or in doses of one-tenth grain, three times daily, by the mouth, it has a more powerful vagotropic action than any other drug, and I regard it as a most efficient cardiotonic and a valuable remedy in hyperthyroidism.[4]

Very recently the advocacy of adrenalin by Gibson, as a drug which has no rival in the management of exophthalmic goitre, fails to harmonize with my theory of this disease. Indeed, there is nothing in Gibson's masterly oration before the recent meeting of the British Medical Association to suggest clearly the mode of action of adrenalin, other than as a hormone regulating glandular activity, yet no one who knows Gibson can question the accuracy of his observations.

Langley has shown that stimulation of the sympathetic roots of the second to the fourth thoracic segments of the cord will cause dilatation of the pupil, protrusion of the eyeball, and acceleration of the heart, yet after the therapeutic employment of a powerful sympathicotropic agent like adrenalin the eyeball recedes, the thyroid gland diminishes, and there is an abatement of other symptoms.

It is not improbable that in this action of adrenalin one is confronted with a condition not unlike that which holds in forced immunity whereby vagus stimulation is activated by adrenalin.

That pioneer in matters therapeutic, Dr. Solomon Solis Cohen, of Philadelphia, advocated the use of adrenalin in exophthalmic goitre more than fifteen years ago. In this communication he likewise refers to the efficacy of thymus preparations alternating with adrenal.

My friend, Dr. W. Blair Bell, of Liverpool, whose researches on calcium metabolism are epoch making, finds that the secretion of the thyroid gland is concerned in promoting the excretion of calcium. He uses calcium for exopthalmic goitre in pregnancy with good results. Calcium salts have been discredited for the reason that they are given in an unabsorbable form. Here is the formula for the "Mistura Calcii lactatis Recentis" of Bell:

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[4] My more recent investigations with *pituitrin* prove it to be a powerful cardiotonic, as determined by the amplitude and duration of the heart-reflex which it evokes.

Pure concentrated lactic acid, 200 grains.

Precipitated calcium carbonate, about 75 grains, to which are added 8 minims of chloroform in 8 ounces of distilled water.

The dose is one and one-half to three ounces every night or every other night. It should be taken on an empty stomach before retiring.

Calcium therapy is often a specific in amenorrhœa associated with headaches, neuralgias, depression, and cardiac weakness. Some forms of menorrhagia also yield quickly to this therapy as well as dysmenorrhœa independent of extrinsic pathological lesions and menopausal disturbances. All headaches and neuralgic pains caused by deficient coagulation of the blood are benefited by calcium salts. Pardon this brief interpolation if the use of calcium therapy is suggested in diseases other than the disease the subject of this paper.

In the employment of my method of treatment in exophthalmic goitre I have confined myself, for experimental reasons only, to stimulation of the vagus by the physiotherapeutic method previously cited, and having established its specific value in treatment I would not hesitate, in the event of failure of the latter, to employ adjuvant measures which increase vagus tone. This refers to the use of pilocarpine, rest, dietetics, avoidance of emotions, etc.

In conclusion, permit me to say that I know of no method of treatment which is more rapid in its results than the method which has been cited for the treatment of exophthalmic goitre. If symptoms are present which suggest atypic forms of hyperthyroidism (*formes frustes*), I should advise a few séances of concussion of the seventh cervical spine not only as a diagnostic but as a therapeutic procedure. Usually the results are immediate in inhibiting tremor, tachycardia, or other signs, provided psychic factors may be excluded attendant on execution of the diagnostic manœuvre. The findings may be gauged more accurately by graphic tracings of the tremor, pulsating struma and pulse. In treatment one must not forget the existence of secondary hyperthyroidism whereby the thyroid may be aroused to augmented secretion by remote foci of excitation. Thus, cases of exophthalmic goitre have been reported where the ablation of adenoids cured the disease.

In my next lecture I hope to demonstrate the specific and therapeutic results achieved in the treatment of aneurisms.

SOME OF THE CLINICAL PHENOMENA ASSOCIATED WITH ARTERIOSCLEROSIS*

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GENTLEMEN: I ask your attention to-day to the records of two patients, the first of whom left the ward for the Convalescent Home a week ago, while the second patient is still under my treatment. Both cases showed marked arterial thickening, a high arterial pressure, and between them they have presented clinical phenomena which are often found associated with such arterial thickening. Later, I shall by means of lantern slides indicate to you my contentions with regard to the changes in the arteries, and the relations between these and the clinical phenomena which have been present, an association which is frequently found to exist in such instances.

To begin with, I shall deal with the clinical phenomena in the two cases.

FAILING MYOCARDIUM WITH THICK ARTERIES AND HIGH ARTERIAL RESISTANCE

CASE I.—James L., laborer, aged 52, was admitted on January 25, 1912. He complained of shortness of breath, palpitation, and headache.

The *history* he gave was that four weeks before admission he noticed that he became very short of breath when at work. His work became such a burden to him that he decided to do nothing for a week. During this week he was short of breath, even on walking upstairs, when hurried, or on going up a slight ascent. As this continued, he sent for a "dispensary doctor," who gave him medicine, but without benefit, so he applied for admission to the Royal Infirmary and was admitted to Ward 29. There was nothing worthy of special note in his previous history: he was a married man with four children, all of whom were alive and well. His habits were satisfactory, and he had a comfortable home.

Circulatory System.—The pulse, on admission, was 60 and regular, but the pulse-wave was small and feeble. The radial arteries were equal, but thickened; the brachial arteries were relatively much thicker, and they gave a pressure of 214 Mm. Hg.

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*A clinical lecture delivered at the Royal Infirmary in Edinburgh, March, 1912.

The *subjective phenomena* elicited were palpitation and shortness of breath; there was no sensation of faintness, but there had been some indefinite pain over the cardiac region.

The *heart* showed its apex to be in the sixth intercostal space, one inch external to the nipple line. The sounds were clear and closed, the second being somewhat ringing in character.

The *respiratory, digestive, and nervous systems* revealed nothing abnormal on examination, save that there was a history of some bowel stiffness.

The *urine* was free of albumin or other abnormal constituent, was normal in amount, and had a specific gravity of 1020.

Treatment.—The patient was kept in bed, put on light diet, and was given tincture of digitalis and iodide of potassium, thrice daily, with magnesium sulphate daily.

On January 30, the report was that he had improved: there was no headache, and he was not nearly so short of breath. The brachial pressure had fallen 30 Mm. Hg to 184 Mm.

On February 3, the pulse was 52 and the arterial pressure 186 Mm. The digitalis was stopped, but the iodide of potassium was continued.

On February 7, the pulse was 70 and the arterial pressure 186 Mm. He felt so much better that he was allowed to get up. There was an X-ray photograph taken of the heart and aorta which showed enlargement of the left ventricle, but no dilatation of the aorta. This examination was made to confirm the opinion that there was no aneurism of the arch of the aorta.

The patient improved so much that he was sent to the Convalescent Home on February 22. During his stay in the hospital he put on about 5½ pounds in weight; the breathlessness and palpitation were recovered from; the pulse was beating between 70 and 80; and the brachial pressure had fallen by 20 Mm. Hg.

Remarks.—In this patient there was presented a group of phenomena which is common, and which it is important you should be able to recognize and to understand. Our patient had been a decent, well-behaved, hard-working man. He had, as is usual in men who have lived a strenuous physical life, greatly thickened arteries in his arms, the brachials being thicker than the radials. This thickening, as I shall demonstrate to you later, is *not atheroma;* it belongs to the group of conditions unfortunately gathered together under the one name of arteriosclerosis. The arterial pressure was high, being over 200 Mm. Hg. The heart was enlarged, but the pulse wave was a poor wave; it did not thump against the artery wall as if every beat threatened to burst it. On slight exertion the patient became breathless, and his heart palpitated: he had no pulmonary or renal condition which could be called on to explain these symptoms, so their explanation had to be found in the circulatory system.

The left ventricle was enlarged, and was no doubt hypertrophied under the stress of a life of physical effort. The muscular coat of his arteries had also hypertrophied, this being the cause of their thickened and hard condition. The balance of the circulation had been maintained easily or, at least, had never been disturbed until shortly before admission to hospital, when it somewhat abruptly became upset, and the patient was rendered unfit for work. Now, when the balance is upset, as it was in this man, it means that the heart has failed for some reason or other: this man's left ventricle failed. A left ventricle may fail in this way from the toxine of influenza or other infective malady; but it may also fail from pure physical fatigue or strain. The 52-year-old heart had probably reached the time-limit of its capacity to respond readily and to rise to any extra effort. The moment that point is reached, and the strain is applied, there is more or less abrupt failure on the part of the myocardium. The result of this myocardial failure was the appearance of the symptoms I have recounted to you.

I ask your special attention to the fact that although this man's brachial pressure was 214 Mm. Hg my house physician decided that digitalis should be given to improve the tone of the myocardium. He judged rightly, but the procedure would have been unreasonable had he taken the 214 Mm. of arterial pressure as the measure of the power of the left ventricle. While thus administering a cardiac tonic, he at the same time gave iodide of potassium internally, for he knows that I regard that drug as a relaxer of arterial hypertonic contraction. I wish you to understand the rationale of these therapeutic measures, for they were the result of a perfectly clear conception of what had gone wrong in the patient's circulation. As already indicated, the left heart had shown symptoms of failure; the arterial pressure at the same time was high, and experience had taught us that one factor in the high reading in these circumstances was a degree of hypertonic contraction of the brachial artery. To meet these two conditions digitalis and iodide of potassium were given together.

Before passing from this conception of failing left ventricle combined with arterial hypertonus I must tell you that the question has sometimes to be solved: is the heart or the arterial condition the primary one? Sometimes it is the one, sometimes the other.

When the heart action becomes feeble the arteries may contract down to adapt their lumen to a lessened volume of arterial blood; while, on the other hand, the arterial constriction may be the cause of the heart embarrassment and failure. It may be difficult sometimes to determine to which the priority ought to be given; while, in some cases, the two factors may so definitely participate in the production of the phenomena that the combined line of treatment will be found to be the most satisfactory. In the particular case under consideration at present we had no doubt that there was myocardial failing. As the myocardium regained tone and power, at the same time the hypertonus disappeared. The thick-walled artery remained, and restoration to health only showed a fall in arterial pressure of about 20 Mm. Hg. We accept 200 Mm., or thereby, as the man's normal pressure with such arteries as he had.

ANGINA PECTORIS; ASTHMA; TEMPORARY HEMIPARESIS AND APHASIA; THICK ARTERIES AND HIGH PRESSURE

Case II.—George L., aged 57, shoemaker, was admitted into the Royal Infirmary on January 25, 1912. He complained of pain over the heart coming on when he began to work or on going upstairs. The pain started in the centre of the chest, went across the heart and down the left arm, and up the left side of the neck to behind the ear. The pain lasted for about half an hour. He also became very short of breath and giddy.

History.—The history he gave was that about 15 months before admission, when he was getting off a tramcar, he was seized with a sharp pain over the heart. The pain lasted about ten minutes, during which time he was unable to move, and found it very difficult to breathe. Since then similar attacks have come on whenever he attempted to work or to make any effort: if he kept quiet they were entirely absent. He has been under treatment by more than one doctor, and has sometimes got a little benefit, at other times no benefit. The attacks had got worse within the two previous months and had lasted longer—usually half an hour.

Family History and Habits.—The family history was satisfactory: he was a married man with healthy children. He had had no illness before the present one. About 18 months ago he began taking whiskey in excess; he used to smoke about 3 ounces of Irish roll weekly. Lately, he has given up taking alcohol, and has reduced his smoking to 1 ounce of light tobacco weekly. He is a well-nourished man, weighing 11 stone 1 pound and measuring 5 feet 4½ inches in height.

Subjective Phenomena of the Circulatory System.—There was no palpitation; but he felt the pain, already referred to, over the precordia, down the left arm, and up the left side of the neck.

Objective Phenomena.—Pulse 88 per minute, regular in time and force, full and hard. Brachial pressure 232 Mm. Hg. Arteries thick and hard and in parts tortuous.

Heart.—Apex in sixth space, 1 inch external to mammary line; on auscultation the first sound at the apex was somewhat faint; there were no murmurs; the aortic second sound was accentuated.

Respiratory System.—There was a slight cough; on auscultation there were some snoring rhonchi, and at the bases some coarse crepitations.

Digestive System.—In this system beyond constipation there was nothing to note.

Urinary System.—No pain or frequency of micturition: the urine was acid, specific gravity 1016; some albumin and a few granular and hyaline casts.

Treatment and Progress.—The patient was kept in bed and put on milk diet. Iodide of potassium and spirit of nitrous ether were given separately, and the bowels were kept freely open with sulphate of magnesia. The pressure came down on the 27th to 202; on the 29th it was 214; on the first of February it was 226; the patient was feeling well, and the amount of albumin had diminished. On February 7, he had the first attack of cardiac pain he had experienced since admission. The pain was first relieved with amylnitrite, and later in the day with nitroglycerin, the pressure falling from 242 to 200 Mm. On the 8th and 9th he was fairly free of pain. On February 10, the pain was absent, but there was considerable dyspnœa, accompanied by impaired chest expansion and wheezing rhonchi on expiration. This was speedily relieved by the administration of stramonium and squill. The arterial pressure for the next few days varied between 213 and 220 Mm.

In the afternoon of February 15, about 4 o'clock, when friends were visiting him, he suddenly felt that he could not speak properly. He felt that he was slurring his words, and he felt his lips wrong on the left side. The arterial pressure was 218 Mm., and one-quarter of a grain of erythrol tetranitrate had no appreciable influence on it. On the following day the left side of the face was definitely paretic and the power of the left hand and arm much diminished. For the next two days, the 17th and 18th, he was given several doses of erythrol, and the pressure varied between 218 and 240 Mm. The pressure was taken frequently and seemed to bear no relation to the taking of the several doses of the erythrol. In the afternoon of the 18th the pressure fell and ranged between 180 and 218 Mm. On February 19, it was noted that speech was better and enunciation clearer, and the grasp of the left hand much increased in power.

From this time onward the speech improved, the paresis of the left side of the face became less, and the power in the left hand steadily increased. The improvement in the paretic manifestations bore no relationship, that could be determined, to the brachial pressure. Between February 20 and March 7 the pressures varied, roughly, between 240 and 265 Mm. During this time he was taking spirit of nitrous ether, and the bowels were kept moving easily.

Remarks.—In this patient there was present the same thickening of arteries as was present in the preceding patient. The brachial pressure was high—230 Mm. Hg—and there was some heart enlargement. There was a difference of five years in their ages; but this man, while the older, was a shoemaker, an occupation not implying arduous physical labor such as our first patient's occupation implied On admission this patient showed albumin in the urine, but there

was no history of previous kidney trouble. The albuminuria may be taken as connected with the arterial thickening and the cardiac enlargement. This is a common association, and I need not enlarge on the cardiovascular changes accompanying granular kidney.

I ask your attention, however, to *three different sets of clinical phenomena* which showed themselves in this man in as many weeks; phenomena which are often associated with the arterial thickening present in this man, but which are not commonly found grouped in the same patient.

The symptom of which he complained when he came to the hospital was clearly *angina pectoris;* he had only one attack after admission, and it was promptly relieved by an active vasodilator; the symptoms disappearing as the arterial pressure fell. The *angina* must, of course, be regarded as an outcome of the cardiovascular and renal condition present: it is prone to occur in such cases when the arteries, from one cause or another, become hypertonically contracted; with the relief of the hypertonus the angina passes off.

The second set of clinical phenomena he presented was *asthmatic:* this was not merely the dyspnœa of a cardiovascular condition, as in our first patient; nor the air-hunger which may occur in uræmia. It was dyspnœa with immobility of chest-wall, and squeaking rhonchi heard on expiration all over the chest. Stramonium quickly relieved the spasm, and with that relief of spasm all abnormal accompaniments disappeared from the lungs. That this true asthma was closely associated with the renal and vascular condition need not be argued.

The third set of phenomena had their origin in the brain and were *paretic* in character. The paresis was most marked in the loss of articulate speech; the left side of the face was also markedly affected; the left arm could be moved and the fingers could be flexed slightly; the loss of power in the left leg was still less, the plantar reflex was normal flexion, but there was ankle-clonus and definite increase of the knee-jerk. The failure of speech came on suddenly, without any uncomfortable feeling in the head or any sense of mental confusion; he simply could not say what he wanted to say—he could not articulate. This motor aphasia was associated with left hemiparesis, and it is interesting to know that this man had trained himself to use the right hand in his work as a shoemaker, and also

to write with this hand. He was, however, born left-handed and still would throw a stone or lift anything heavy with that hand. Notwithstanding this education of the left side of his brain, the law remained that, being born left-handed, a left hemiparesis carried speech paresis with it. Let me ask your attention to this abrupt onset of speech failure with paresis of face, arm, and leg. In my last clinical lecture I spoke to you about the clauses of hemiparesis and hemiplegia, and you will remember that to hæmorrhage, softening from thrombus or embolus, I added, what you will find in none of the text-books, namely, *angiospasm*. This last is probably the factor which acted in this case. The reasons which point to this explanation as being the right one are: (1.) The paresis was extensive and yet was not accompanied by any subjective mental phenomena as giddiness, stupidity, or any failure to realize what had happened: he quite vigorously indicated how he suddenly was conscious that he could not speak and that he felt he could not move the left side of his mouth. (2.) In my experience a hæmorrhage would have been accompanied with definite mental obfuscation, if not with stupor or temporary unconsciousness. (3.) There was no cardiac lesion, so that embolism had not even the existence of an endocardial murmur to suggest it as a possibility. (4.) Thrombosis is a permanent condition, and is presumably always followed by some extent of softening, and the persistence, therefore, of some paresis. (5.) In angiospasm the symptoms, as in this case, soon begin to lessen. If some symptoms persist it means that localized softening has resulted, and the softening may follow on the anæmia due to the angiospasm, or the formation of a localized thrombus has been favored, followed, of course, by softening of the area to which blood should have been supplied by the blocked vessel.

As to the determination of the cause in this patient the choice seems to me to lie between thrombosis or angiospasm, with possible thrombosis as a consequence of the latter. Sudden onset, as sudden as in this case, is against the view of a primary thrombosis, and favors the view of angiospasm.

With regard to the site of the angiospasm, one tends to think of it as occurring in the motor pathway, and not in the cortex; for, in the cases where some permanent paresis persists, the lesion is found in the tract and not in the gray matter. The question why

angiospasm occurs in the pathway has the same answer as the question of the occurrence of hæmorrhage and softening in the same region. We know too little about the conditions which affect the cerebral circulation to presume to answer many questions which present themselves for solution. We may, however, safely say that the motor pathway is the commonest seat of lesion as surely as it is the most constantly employed and most constantly traversed road in the brain.

Manifestations such as were present in this patient, and which were assuredly to be referred to vessel conditions in the brain, are not uncommon in patients with arteries such as this man had. Other cerebral phenomena are met with in the same class of case, and are due, presumably, to corresponding vessel conditions; but I do not deal with these now. I must add to this, however, that in the considerable experience I have had of the brain conditions in persons with arteriosclerosis I have usually found that cerebral manifestations occur along with a hypertonic contraction of the systemic arteries as judged by the rise in brachial pressure; and, conversely, that the symptoms disappear with the relaxation of the hypertonus. This association has been very marked in many cases; indeed, the control and removal by therapeutic measures of the hypertonus have usually led to prompt relief of the symptoms. In this particular patient it was not clear that we effected any therapeutic benefit by the remedies used. We probably in some measure controlled the arterial pressure, but we were so uncertain of any beneficial effect being produced that the use of the more powerful vasodilators was stopped; and, although thereafter the pressure rose, the rise was not associated with any worsening of the paresis, which still remained. When the cerebral symptoms vary with the changes in the systemic circulation we easily assume that the one circulation varies in harmony with the other; but, as in this case, it is sometimes found that, even when you have affected the systemic circulation, there is no evidence of the cerebral having been similarly influenced. This is a matter regarding which only inferences can be drawn from the symptoms; but the inferences may be supported by the time and mode of disappearance of the symptoms. In this case the view that the paresis was due to local angiospasm is, I think, strongly supported by the fact that by the middle of March the paresis had disappeared

and the power of speech had entirely returned. Had there been destruction from hæmorrhage or softening, such a result could hardly have been attained.

General Considerations.—I have in these two cases presented to you a record of three sets of clinical phenomena, and, as I have already stated, the phenomena occurred in patients with thickened arteries giving high hæmomanometer readings. In one patient there was albumin in the urine, in the other there was none. I ask your attention to the thickened arteries present in both the patients. Such arteries are commonly referred to as *arteriosclerotic*. This term was introduced from Germany, and a nodular and a diffuse variety was spoken of and written of; but if you go into German and English literature you will find that, when the structural changes are given in detail, what is described is the atheroma which we knew about before the word arteriosclerosis had been coined.

In 1901 I drew attention to many observations I had made on thick radial arteries I had felt and seen during life. I found that most of such thickened radial arteries were not atheromatous, but were thickened by hypertrophy of their muscular coat. I suggested that, as the word arteriosclerosis was in general use, it would be an advantage were it confined to indicate this kind of thickening, while the word atheroma could be confined to the condition to which it was originally given. Pathologists and physicians have been to a large extent deaf to these representations, so that the absurd confusion resulting from the use of one word for totally different conditions remains and will remain until the authorities decide that this state of things must end. Meanwhile I can show you the result of my observations, and I doubt not that at the conclusion you will wonder why the pathologists do not without more delay accept and teach what is of very considerable clinical importance. I shall first show on the screen a normal radial artery. I next show you radial arteries which are atheromatous, and some which have calcareous infiltration of parts of the middle coat. In these atheromatous and calcareous radials I point out to you that the muscular coat at the part where the vessel is free from these changes is thickened and hypertrophied. I next show you a number of radial arteries which had been recognized as thickened during life, and in which there is no atheroma, but in which there is marked thickening of the

muscular coat. This is the common condition, and it may be extensive without any atheroma from the radial back to the aortic arch. It is to this condition that I have proposed the term arteriosclerosis ought to be confined. I next show a normal brachial artery, and I ask you to note the extraordinary difference between it and brachial arteries thickened in the way I have indicated. You readily note the difference in the thickness of the wall; and you see that you are dealing with a pure hypertrophy of the muscular coat, and no atheroma.

I am not now to discuss with you how the muscular coat hypertrophies. I ask you meanwhile to recognize the absurdity of using the same word for arteries like these as is applied to atheroma.

I next ask you to realize that such thickened arteries add, and must add, to the amount of pressure required from the armlet of the hæmomanometer to stop the pulse-wave. But I ask you still further to realize that when from any cause such an artery undergoes hypertonic contraction the wall is much thickened and the lumen is much diminished and the resistance to compression by the armlet is greatly added to. It thus follows that when you take the brachial pressure in such cases as I have presented to you, you must keep in mind these factors, namely: (1) blood-pressure; (2) structural change in the artery wall; and (3) hypertonus of the muscle coat of the wall.

The sphygmomanometer or hæmomanometer is a very useful clinical instrument, but in the two cases we have been considering to-day it has not been as useful as it often is, I might even say, as useful as I usually find it to be.

THE DIAGNOSIS AND TREATMENT OF OBSCURE CASES OF PYELITIS AND HYDRONEPHROSIS

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The study of kidney conditions has made such progress in the past decade that the specialist is now rarely called in to see a well-marked case of pyelitis or advanced case of hydronephrosis which has not been previously recognized by the general practitioner.

But many cases of pyelitis and pyelonephritis are overlooked, and even recover spontaneously. Many cases, however, are not diagnosed and they continue in ill health.

Taking it for granted that the physican has the advantage of frequent consultations with colleagues, attendance on medical society meetings, and opportunities to come in contact with the vast clinical material of well-managed clinics, and is therefore familiar with the advanced lesions of the kidney pelvis, I wish to present for discussion a few observations that may not be so generally recognized.

In a recent paper on pyelitis the author [1] classified his cases occurring up to that time under the following etiological headings: cases due to congenital malformation, exposure to cold, associated with interstitial nephritis, following gonorrhœal infection, following typhoid fever, following operation, associated with cystitis, with gastro-intestinal disorders, due to appendicitis, associated with stricture, pyelitis of pregnancy, and pyelitis of the puerperium. This forms a fairly complete classification of the possible causes of pyelitis, and I shall cite a few cases to illustrate some of its more important etiological factors.

Case I.—Mrs. M., on her way from St. Louis to Atlantic City in search of health, was seen by Dr. J. A. Chatard, of Baltimore, and because of her past history she was referred to me in March, 1912, to find whether she might be having trouble with her right kidney.

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[1] Hunner, "The Treatment of Pyelitis," *Surgery, Gynæcology and Obstetrics*, xv, 444, 1912.

She was 36 years of age, and the mother of three children, the youngest aged 6. She had been operated upon for a perineal repair two years previously, and apparently had a perfect convalescence until the fifteenth day, when she was placed upright in bed and her temperature rose to between 103° and 104° F. For a day or two before this she had much pain and burning in the bladder region on voiding.

For eight months after leaving the hospital the patient had repeated attacks of chills and fever at intervals of one week to one month. The blood had been examined for malaria, and bladder cultures had been taken, and vaccines prepared from a bacterial growth had been used for about a half a dozen injections.

For 16 months before I saw the patient she had been free from the attacks of chills and fever, and at the time of her visit she was not complaining of bladder symptoms or of pain in the kidney region. Her chief complaint was of general malaise and of the fact that she had spent two years in travelling and in sanitaria in a fruitless search for health.

A specimen of urine catheterized from the bladder and examined within a half hour showed many motile short rod bacilli occurring, singly, in pairs, and in chains of three. There were a few pus-cells and a few epithelial cells. There were no casts and the urine was free from albumin.

On the patient's second visit I did cystoscopy, and catheterized the right kidney. The bladder appeared normal; each ureteral orifice was somewhat red and swollen-looking. The right kidney pelvis injected to the point of discomfort held 9 c.c. The urine from the right kidney showed about the same fine turbidity as the bladder specimen, and this was found to be due to a thick invasion by the colon bacillus. There were no leucocytes in the kidney specimen. The kidney was washed with 60 c.c. of 1 to 1,000 silver nitrate solution, and five hours after this treatment the patient had a severe chill and a fever of almost 103° F. She was naturally very miserable, but expressed herself as being very happy that the possible cause of her ill health had been discovered, for she was certain that this attack was similar to those she had suffered for eight months after operation.

The patient came to my office four days later and had a similar treatment with similar results, the fever coming on in two hours after the treatment. At this visit the urine catheterized from the kidney had a thick milky turbidity and was loaded with bacteria, epithelial cells, and many cells which on staining showed a double nucleus, and I could not determine whether these were lymphocytes or dividing epithelial cells. There were a few ordinary appearing polymorphonuclear leucocytes.

This was the first experience I ever had in producing acute pyelitis chills after renal catheterization. I considered that the reaction might possibly arise because of some point of narrowing in the ureter, the lumen becoming temporarily closed because of swelling of the mucosa due to the trauma of catheterization. I therefore sent the patient to the hospital, and on the two succeeding kidney irrigations I dilated the ureter by means of a wax bulb on my renal catheter. After the first of these dilatations the patient had a chill and a temperature of 103° F. within one hour of the kidney washing. Five hours after the washing the temperature was 105° F. After the second dilatation with a larger wax bulb there was no reaction.

The patient had five irrigations of the kidney with silver nitrate solution 1 to 1,000, and in spite of the acute reaction after the first three irrigations

the catheterized specimen from the kidney showed negative culture results after the fourth and fifth irrigations. The bladder culture was negative after the fifth irrigation.

Many cases are wrongly diagnosed and treated as some other disease, particularly as cystitis, malaria, and typhoid fever. In my experience the puerperal kidney infections are particularly likely to be treated as cystitis cases because in these the bladder symptoms are often prominent, and the pain in the back and temperature reaction may not be marked.

Case II.—In the paper just referred to, I reported at length the case of a child, 10 years of age, whom I saw in the fall of two successive years, suffering first with pyelitis of the left kidney, and the following year with pyelitis of the right kidney, due to congenital malformation. During each year the child's long-continued illness had been diagnosed as typhoid fever. On her second visit her temperature chart was brought with her to show that she actually had had typhoid fever. This chart shows that for five days in August her temperature ranged between 102° and 104.5° F. and then within two days fell to approximately normal, where it remained for two weeks, and then for one day reached 103° F., again reaching normal within forty-eight hours, and after four days going to 103° F. on two successive days.

On each visit the child failed to show a Widal reaction, and a pure culture of the colon bacillus was grown from the respective kidneys. On each visit the infection was eradicated after five washings with silver nitrate solution, 1 to 3,000 being used for the left kidney, and a year later 1 to 1,000 being used for the right kidney.

In an important group of cases the pyelitis is diagnosed and treated as such when the pyelitis is secondary to appendicitis. In 1908, I had the pleasure of presenting a paper before the College of Physicians of Pittsburgh,[2] entitled "Acute Pyelitis due to Acute Appendicitis." I reported four cases in that paper, and have since seen two well-marked cases. Others have been reported in the literature since that paper.

The great significance of this group of cases lies in the fact that the urine examination, upon which we are so dependent in dealing with many obscure abdominal conditions, may be the chief factor in leading to an error in diagnosis. The pyelitis case is rarely in immediate danger, while a few hours' delay in the acute appendix case might determine the death of the patient.

[2] *J. A. M. A.*, vol. l, p. 1328.

By simply bearing in mind the possibility of these two conditions occurring together, there should be no great difficulty in diagnosing the appendicitis, even when the high fever, chills, and urine examination point to a positive involvement of the kidney. In a recent case sent to me for pyelitis of pregnancy I found that the patient was suffering from this condition, but that she also had distinct evidences of acute appendicitis, so I operated at once, finding an acutely inflamed appendix and considerable involvement of the peritoneum about the appendix in the presence of a five months' pregnancy. The pyelitis yielded to the usual expectant plan of treatment given in such cases.

Pyelitis and various degrees of hydronephrosis secondary to ureter stone, and particularly those secondary to stricture of the ureter, are frequently undiagnosed, or if diagnosed, the treatment is aimed at the kidney condition rather than at the primary cause.

As an illustration of a patient suffering with hydronephrosis due to ureter stricture, following the passage of a stone from the ureter, I wish to cite the following:

Case III.—Mrs. H., aged 47, mother of two children, aged 19 and 7 years. The patient was sent to Dr. L. F. Barker, a diagnosis having been made of probable tuberculosis of the kidney. She had passed a stone from the left ureter about five years ago. Since that time she had had more or less pain in the left kidney region and thought it was trouble due to gas in the bowels.

The diagnosis of tuberculosis of the kidney must have been made by finding smegma bacilli in a voided specimen, for on catheterizing urine from the bladder it was quite without pathological elements. On catheterizing the left ureter with a wax-tipped bougie, this was permanently obstructed at about the pelvic brim. I transferred the patient to the X-ray room and injected 10 per cent. collargol to the point of pain in the kidney region. The X-ray picture shows a definite dilatation of the kidney pelvis and of the ureter down to the pelvic brim. The dilated pelvis and ureter held 28 c.c. A few days later I had no difficulty in passing a No. 5 pointed renal catheter past the obstruction, the catheter carrying with it a wax bulb for dilating the stricture. These dilatations were carried out for several treatments, when the capacity of the kidney pelvis was reduced from 28 c.c. to 8 c.c. Now, five months after the first treatment, the patient is quite free from her original pain.

The two following cases illustrate in a striking manner the insidious character of stricture of the ureter, and emphasize the importance of looking for this condition in dealing with certain kidney lesions, the etiology of which is obscure:

Case IV.—Miss B., aged 45, referred in July, 1912, by Dr. J. A. Chatard. The patient had been suffering for about one week with all the signs and symptoms of an acute pyelitis. The attack began with acute pain in the left kidney region, followed four days later with pain in the right kidney region. She had had repeated chills and a hectic fever ranging as high as 104° F. The suffering from backache and soreness in both kidney regions had been intense, and she was considerably worried by the frequency of voiding.

The patient, under Dr. T. R. Brown's care, had had three similar attacks in 1909 and 1910, the pyelitis symptoms in these attacks being limited to the right side. In each of these attacks Dr. Brown kept the patient in bed from one to two weeks, forced water drinking, and gave large doses of hexamethylenamine, with prompt results as to apparent perfect recovery. After the third attack, in May, 1910, the patient spent some time at Bedford Springs, and she considered herself perfectly well up to the attack of double pyelitis in which I was called to see her.

Dr. Chatard had been treating the patient for double pyelitis, but as she failed to improve I was called in consultation and we sent her to the hospital. The patient had a palpable and very tender mass in each kidney region. The urine showed one-third of its volume as containing pus. On cystoscopy the bladder looked approximately normal, and each ureteral orifice looked normal except for the small size. On passing renal catheters it was found that both ureters were infiltrated and narrow at the bladder orifice. In addition, the right ureter was found to have two stricture rings above the orifice and within about 5 cm. of the bladder. A steady stream of urine ran from each kidney, showing a double hydronephrosis. The urine from the right side was very turbid and showed a considerable amount of pus, while the left kidney yielded a urine of almost milky turbidity. This side might well have been designated a pyonephrosis. The right kidney was found to have a capacity of 100 c.c., and the left kidney of 120 c.c.

Treatment by pelvic lavage with silver nitrate 1: 500 was begun at once and kept up twice a week for about three weeks in the hospital. The patient was then well enough to leave for home, and has since been coming to my office for treatments once or twice a week. The right kidney cleared up remarkably after the first two irrigations, the urine appearing amber clear to the naked eye, but showing a considerable deposit of pus and bacteria on centrifugalization. The infection in the left side seemed much more persistent, but steadily improved. On October 15 I made the following notes: Both sides catheterized with whistle tipped No. 8 catheters, prepared with a wax bulb about 5 cm. back of the tip of the catheter. There was slight obstruction on the left side just after entering the ureter, and on the right side three points of obstruction could be detected, one just after entering the ureter, and two within four or five cm. of the bladder. The urine from either side looks absolutely clear, but a colon bacillus culture is still obtained, and microscopically a few pus-cells are found in the urine from either side. Dilating each kidney pelvis to the point of discomfort, the right side holds 60 c.c., and the left side 35 c.c. It will thus be seen that the left side, which held 120 c.c. and was the seat of the most severe infection at the beginning, has contracted down toward the normal size much more than the right side, which at first held 100 c.c. This agrees with what we might expect theoretically from the fact that the previous attacks have all been

on the right side, thus leaving the pelvic tissues more infiltrated and less contractile than on the left side.

I think it is safe to assume that the patient has had the ureter strictures from some period before her first pyelitis attack on the right side, and that the bilateral hydronephrosis has been developing over a long period in spite of her apparent good health.

The sulphophenolnephthalin test at my first examination in July yielded in the first hour, after a deep gluteal injection, 19 per cent. from the left kidney and 15 per cent. from the right. At the last examination, October 15, the left kidney showed 17 per cent. and the right 14 per cent. Again we see, in spite of an apparent worse condition of the left kidney, that the functional value is higher than the right. I do not attempt to explain why the percentages of coloring matter thrown out during the acute inflammatory stage in July, when the kidneys were under high pressure from the hydronephrosis, should be greater than the percentages thrown out recently, after all signs of acute inflammation have subsided. These readings were made by two different laboratory men, and I am inclined to ascribe the difference in readings to this fact.

The findings in this case would suggest the advisability of a thorough investigation of the urinary tract of any patient who has repeated pyelitis attacks, in spite of the fact that they promptly clear up on medical treatment.

Case V.—Miss O., aged 27, came to my cystoscopy dispensary at the Johns Hopkins Hospital on October 1, 1912. She complained of a dull aching pain in the left kidney region for the past year. This pain varied in severity, but was never cramp-like. It was not increased at the menstrual period. She never noticed bladder trouble of any kind. She never had rectal tenesmus. On palpation the lower pole of the left kidney could be felt. This seemed in normal position. There was no apparent enlargement, and no particular tenderness. Urine catheterized from the bladder was normal. On cystoscopy, the bladder appeared normal. The left ureter orifice was rather red and swollen, standing out prominently. A No. 5 renal catheter with pointed tip was prepared with wax tip and wax rings, to test for ureter stone. The catheter obstructed about 6 to 8 cm. above the bladder and doubled back in the bladder. The stilette was reintroduced and the catheter again met the obstruction, but was pushed by and on to the kidney. The urine caught for examination was bloody (due to trauma), but showed no other pathological condition. The kidney was injected until the patient complained of a "cramped feeling." The patient said this cramp-like pain was more toward the median line of the abdomen, and more acute than her former pains, which were always dull in character and located in the posterior kidney region. However, after the patient was dressed and ready to go home, she volunteered that she felt as if her old trouble had been stirred up.

The capacity of the kidney pelvis was 25 c.c., thus showing a definite hydronephrosis. The wax tip and wax rings were not scratched, thus showing that the obstruction in the ureter was not due to stone.

Late that night Dr. A. L. Wilkinson called me up to ask what I had done

for the patient, as he had just been called to see her and was forced to give a hypodermic of morphine for her agonizing pain. I have seen the patient twice since the first treatment, and by using larger wax bulbs have been able to dilate the stricture so that she does not have closure of the ureter after the treatments. Dr. Wilkinson gave me valuable additional notes in her case which show that he had been unusually careful in attempting to make a diagnosis, but had fallen short in the one step necessary to clear up the diagnosis and to bring about relief. He had first seen the patient about two years ago, when, after a brief investigation, he decided that she had lumbago. A year later the patient returned to him complaining of the more definite dragging pain in the left kidney region, and he then thought that she might have stone in the kidney or ureter. Several examinations of the urine were negative, and he then sent her to Dr. Ashbury, who took two excellent plates of the urinary tract region with negative results.

There has been considerable discussion in the literature as to what degree of distention the kidney pelvis must obtain before it is worthy of being called a hydronephrosis. Of course, this involves the primary question as to the content of the normal kidney pelvis. My observations have been confined entirely to the kidneys of women, and, using the Kelly method of cystoscopy, I have had the advantage of being able to use a large catheter for these tests, and usually add the wax bulb a few centimetres back of the tip of the catheter in order to prevent any reflux of urine. I am careful to warn the patient beforehand not to wait for a sharp pain, but to let me know as soon as she feels a discomfort in the side. I measure the content of the pelvis, not by the amount of the injection, but by the reflux that comes in a steady stream through the catheter.

In most normal cases, this method yields 7½ c.c. In large women, I am not surprised to get 8 and 9 c.c., and exceptionally 10 c.c., as the content of the kidney pelvis.

I have always agreed with Dr. Howard A. Kelly, in this controversy as to what we shall call a hydronephrosis, that any enlargement of the kidney pelvis causing symptoms should be considered as a pathological condition, and for this condition the best term in common use at present is hydronephrosis.

To my mind the best proof that we are dealing with a pathological condition in these low-grade enlargements of the kidney pelvis is the repeated observation that I have made in cases due to obstruction by a stone or stricture in the ureter, that the kidney pelvis invariably shrinks from its dilated size down to the normal,

or approximately normal, size soon after the ureter obstruction is removed. In the excessive dilatation of the kidney, by long-continued ureter obstruction, the shrinkage is often marked, but, of course, the final capacity does not approximate the normal.

It is difficult, and often impossible, to settle upon the etiological factor in these cases of ureter stricture. I have already described in Case III the occurrence of ureter stricture succeeding a ureter stone. I have seen local tuberculosis of the ureter following kidney tuberculosis, although in this disease the ureter inflammation is usually general. Knowing what we do of the effects of a gonorrhœal infection on the urethral and para-urethral tissues, one would expect to frequently find periureteral infiltration following gonorrhœal cystitis. One of the common sequences of gonorrhœal cystitis in women is an infiltration of the mucosa about the ureteral orifice reducing the orifice from a slit-like opening to a very fine, almost pin-point, round opening. I have seen many of these infiltrated, small, round ureter openings, which interfered with easy catheterization, but I have never seen one which seemed to interfere with the ureter function of freely carrying down the urine. For a number of years I have been expecting to find periureteral thickening at a distance from the ureter opening in these cases of former gonorrhœa of the bladder. I made such an observation for the first and only time this past summer in the case of one of my dispensary patients, a young colored woman who had every evidence of a past gonorrhœal infection, such as stricture of the urethra, chronic urethritis, trigonitis, narrowing of the ureteral orifices, and, in addition, an annular stricture of the ureter situated two or three centimetres above the bladder.

Of course, we get ureter obstruction in many cases of carcinoma of the cervix. Obstruction may likewise come from pelvic inflammatory disease, pressing upon the ureter or dragging it out of place. It is conceivable that appendicitis or other inflammatory conditions along the upper ureter might have the same effects. Pelvic or abdominal tumors, and downward displacements of the pelvic viscera sometimes result in obstruction to the ureter.

If we now return to the consideration of ureter stricture we find, on going over the literature, that those cases for which there is no evident cause are classified as congenital stricture of the ureter.

Agreeing that some of the cases have a congenital origin I am firmly convinced that too many have been thus classified.

In a paper [3] entitled "Chronic Urethritis and Chronic Ureteritis Caused by Tonsillitis," which I read before the Southern Surgical and Gynæcological Association at the meeting in Nashville, December, 1910, I called attention to the probability of some of our obscure ureter stricture cases being due to infections or toxins derived from the tonsils. My observations since that time have served to strengthen this view, and to make me feel certain that not only tonsillar infections but the various infections of the cranial sinuses may result in damage to the distant urinary organs.

I reported two cases in this first paper, and in my more recent paper on pyelitis, read at Washington last December, I added two other cases of ureter stricture which I considered due to tonsillitis. Of eleven cases of stricture dealt with since last December, I consider that three belong to the tonsillar group, and that one was due to gonorrhœa. The etiology was quite obscure in two, and five were associated with stone, either in the ureter, kidney, or bladder.

The association of the so-called rheumatic diathesis with stone in the urinary system is a time-honored one, probably familiar to all of you. The knowledge of the relationship between tonsillitis and rheumatism is of later development. We know that acute pyelonephritis and acute prostatitis follow acute attacks of tonsillitis, and it seems quite reasonable to believe that some cases of stone in the urinary tract may be traced back to infections from the tonsils or the sinuses.

TREATMENT

After what has been said in my report of cases, I need not say much under the heading of treatment. Many cases of acute pyelitis recover on the proper expectant treatment. This should include rest in bed, liquid diet, the forcing of fluid, and counter-irritation over the kidney region, or at least the keeping of this region of the skin surface under conditions of equable temperature. To these hygienic measures may be added the administration of hexamethylenamine, although the action of this drug is very uncertain, some patients not secreting enough formalin to be of antiseptic value, even

[3] *J. A. M. A.*, vol. lvi, p. 937.

with very large doses of the drug. In systematic experiments, while interne at the Johns Hopkins Hospital, some twelve to fifteen years ago, I found that some urinary tract infections cleared up very promptly with the administration of urotropine, and that others were benefited very little or not at all. Since that time I have taught my students that if the hexamethylenamine was not effectual in the first week or ten days its administration might better be discontinued. More recently it has been found that a patient who does not secrete enough formalin on the usual dose of 10 to 20 grains three times a day may secrete antiseptic quantities if the dosage be greatly increased. As much as 200 grains a day may be given, if the effect on the urinary tract is closely watched, as the toxic effects first appear in the kidney.

If a patient fails to respond to the hygienic and medicinal measures above outlined, recourse should be had to the lavage treatment already described in my report of cases. It is self-evident that any ureter obstruction must be overcome before we can offer permanent benefit to these cases of infected kidney.

I have not yet had a case of colon bacillus pyelitis that has failed to respond promptly to the pelvic lavage with silver nitrate solution. One patient with a puerperal pyelitis of four years' standing had a negative culture after eight washings of the kidney. This patient was all but bedridden because of her kidney condition, and after three weeks' treatment she was able to take long walks. This was the greatest number of treatments I have had to give with the silver nitrate in any case except the one related above (Case IV) who had the bilateral ureter stricture and marked hydronephrosis.

Of my so-called tonsillitis cases of stricture of the ureter, two have had infected urine, and it is significant that both of these infections have been by the staphylococcus aureus. I have purposely persisted for several months in the lavage of these two cases with silver nitrate solution 1 to 500 in order to test the efficacy of this drug in dealing with the staphylococcus. I am now satisfied that we must seek for something better in these cases. Koll,[4] of the Michael Reese Hospital, Chicago, has reported successes in treating both colon bacillus and staphylococcus aureus cases by washing the pelvis with 2 per cent. solution of aluminum acetate. My colleague.

[4] *Transactions of the American Urological Association*, 1911, v, 94.

Dr. C. F. Burnham, informs me that he had had successes in two or three staphylococcus infections by using formalin solution 1 to 2000, taking the commercial solution as a standard. I have begun treating my two staphylococcus cases with the formalin, and if not successful with this I mean to try the aluminum acetate.

SUMMARY

If I were asked to summarize the foregoing remarks, I should say that they attempt to formulate a plea for more care in the diagnosis of obscure abdominal complaints.

(1) A careful urine examination is a *sine qua non*. In women time should not be wasted on the examination of a voided specimen if there is reason to suspect disease of the urinary tract located above the urethra.

(2) Leucocytes or red blood-corpuscles in a specimen catheterized from the bladder indicate an abnormality the origin of which should be diligently sought.

(3) A bacteriuria, even if unaccompanied by pus, blood, or albumin, or if associated with only a few leucocytes, may mean a kidney lesion the importance of which to the patient's health may be far greater than is indicated by the changes in the urine.

(4) In a case of suspected pyelitis or intermittent hydronephrosis, repeated urine examinations are indicated, for the urine may be quite normal or show only a mild bacteriuria during the acute attack due to blocking of the affected kidney or the infected calyx. In the hydronephrosis patient the urine may be normal at all times.

(5) In the case of hydronephrosis do not be satisfied in making a diagnosis after the kidney has become an easily palpated tumor mass.

By this time your patient has probably had at least one operation for the removal of a normal appendix, and she may have had one or two explorations for stomach ulcer or gall-stones. She will almost certainly have had several rest cures for neurasthenia.

Surgery at this late stage means the sacrifice of an organ that should have been saved, or the patching up of a kidney so badly damaged that it is a question whether a complete removal would not be the more conservative operation.

The plea that you do not use the cystoscope and have no

facilities for scientific X-ray work does not justify you in allowing your patient to suffer until the kidney lesion becomes so plain that a veterinary surgeon could make the diagnosis.

There lives just around the corner from you a young colleague who is spending long hours at the painstaking and accurate work of cystoscopy, and he is closely affiliated with one or more men who are making sacrifices because of the wonders they can bring to light by means of Röntgenology.

You may think lightly of their accomplishments and may deprecate their enthusiastic claims. Would it not be better to seek their co-operation and contribute your calmer judgment obtained at the bedside or the operating table in helping to correct possible misinterpretations due to their enthusiasm and lack of contact with actual conditions?

How often we hear the veteran claim that he could have given you the same diagnostic picture merely by getting the patient's history and making the usual physical examination! Let us not forget that this applies only to the far-advanced case.

Our plea is for the team-work that will insure to the patient the earliest diagnosis and the simplest treatment commensurate with our newer methods of precision.

PYELITIS: ITS CLINICAL VARIETIES AND TREATMENT

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Pyelitis is a disease to which scanty reference is, as a rule, made in text-books. It is, however, a disease which is daily coming more into prominence, as it is becoming recognized that it is one of fairly common occurrence. In this paper I have endeavored to group the disease into its clinical varieties; and this grouping will, I trust, for want of a better classification, serve a useful purpose. At the same time, in using the term pyelitis I have chiefly concerned myself with those cases of the disease which are due to infection with the bacillus coli, or, at any rate, with one of that class of organisms. Such are the varieties which I have named infantile and puerperal. These are specially amenable to medical treatment, and consequently belong to the domain of the physician rather than to that of the surgeon. I have, therefore, made special reference to these varieties, and have only mentioned the other varieties.

Before proceeding to refer to the various clinical groups of pyelitis I may remark that for some time I have felt that in our examination of the urine we do not employ the microscope and the culture-tube so frequently as we might. Apart from finding colon bacilli, we may, by these means, detect the presence of Streptococci which are now known to exist in the urine in certain forms of rheumatism (?) and in some cases of gastric (?) affection, tonsillitis, eczema, as well as in other conditions. In fact, I am more and more convinced that we have still a wide field before us in the bacteriological study of the urine in disease. Workers in this field are at present but pioneers, and consequently our knowledge of the bacteriology of the urine is very scanty, but I feel that the time is not far distant when a bacteriological examination of the urine will be regarded as of even greater importance than the ordinary chemical

investigation, the value of which has been fully recognized. The more we study pyelitis, at all events, the more will we recognize the absolute necessity of keeping in view the presence of micro-organisms in the urine, and of regarding these as a possible source of the symptoms in what must otherwise prove to be very obscure cases.

I shall now take up the various clinical groups of pyelitis, referring only to those points in each regarding which I have had some personal experience. In this way I hope to be able to add a little to our knowledge of a disease which must very soon receive more attention at the hands of medical writers than it has done up to the present.

INFANTILE PYELITIS

This variety is most commonly met with in quite young infants, but may also occur in childhood. In some cases the condition is due to renal calculi or tumors, to tuberculosis of the kidney, or to one of the infectious fevers. In most instances, however, infantile pyelitis is due to infection with the colon bacillus. The condition may or may not be associated with cystitis. Females are much more frequently affected than males, and the majority of cases occur before the age of two years. It is alleged that females suffer more than males because of the greater readiness with which the bacilli gain an entrance into the urinary tract in the female. I am inclined to question this mode of infection however, and look upon these cases as arising by extension directly from the intestinal wall to the kidney, or, it may be to the bladder primarily. The possibility of a blood infection must not be lost sight of, as in all probability some cases of infantile pyelitis owe their origin to this source. There is no doubt that in many cases there co-exists an acute cystitis, but the symptoms of the latter are either extremely mild or they are masked by those of the associated pyelitis. The fact that there is no involvement of the genital tract in the female rather militates against the spread of infection from the rectum to the urethra.

A typical case of infantile pyelitis presents the following features: The infant—usually a female—has been perfectly healthy, but in some cases has had a pre-existing attack of gastro-enteritis. Constipation is often present. Quite suddenly the patient is noticed to become pale and has a fit of shivering. The temperature rises to

104° F. The patient cries when it attempts to micturate, which it does frequently, and passes a small quantity of cloudy urine at a time. The infant soon becomes very cross, fretful, and irritable, sleeping badly and refusing the breast or bottle. The temperature continues to rise and fall. The patient, as time goes on, is apt to become emaciated and anæmic-looking. It may even develop cerebral manifestations, and may become listless and somnolent. Examination of the urine will reveal the presence of pus with a small amount of albumin. Motile bacilli will be readily detected under the microscope. The urine has a markedly acid reaction and usually a characteristically disagreeable odor. The pain on attempting to pass urine will be more marked if cystitis is present as well, and micturition will usually be more frequent than is the case when pyelitis exists alone. In some cases, but by no means in all, there is considerable pain on pressure over the region of one or both kidneys. It is a noteworthy feature that the temperature is often lower when abundance of pus is discharged than when the latter is scanty in amount. In other words, the better the drainage the more likelihood is there of a lowering of the temperature taking place.

As a general rule, these cases recover under treatment, although it may be weeks, or even months, before the urine becomes absolutely normal. It must not be supposed, however, that death never takes place. Thus the patient may die from exhaustion and the long continued fever, or a nephritis may supervene which may prove fatal. The earlier the diagnosis is made and suitable treatment is undertaken the better is the prognosis.

The first step in the treatment is to secure a regular evacuation of the bowels by some such drug as *hydrarg, subchlor,* or *hydrarg. c. creta.* The second step is to render the urine alkaline by means of *potassii citras* in doses of 0.3 gramme every two or three hours. It is merely a waste of time to try other drugs in this condition when it occurs in infants. Urotropine, in my experience, is worse than useless, and may actually lead to the onset of nephritis when given to young infants. Salol I have never employed, as it is not a safe drug to administer in the case of young patients, owing to its irritating effects upon the kidney. Besides, it seems unnecessary to experiment with other vaunted remedies when we have a drug, namely, *potassii citras,* which usually acts so promptly and bene-

ficially in these cases. The use of vaccines has also been suggested, but these also appear to be unnecessary, save, perhaps, in desperate or very chronic cases.

As a matter of fact, the treatment of infantile pyelitis is not usually difficult, provided the case is seen early and the diagnosis promptly made. If, however, for any reason there is delay in applying the suitable remedy, then all kinds of unfortunate circumstances may arise. In the very worst cases the patient may die. Death may be due, as has been already mentioned, to asthenia, or to exhaustion from the long continued temperature, while an extension of inflammation to the kidney substance may bring about a fatal issue. In one case of very severe pyelitis, the patient, a male of two years, died from bronchopneumonia, which set in during the fifth week.

As everything depends on the diagnosis in these cases, it is of the utmost importance that a very thorough examination of the urine should be undertaken. I have constantly urged that in our examination of infants and children far too little attention is paid to the urine. In adult cases we often examine the urine as a matter of routine, and in hospital practice we never neglect to do so. Not so in the case of children. The reason may be given that it is not so easy to obtain specimens in the case of young patients. This, however, is really a very lame excuse. In fact, it has no foundation, for with a very little trouble it is quite an easy matter to get a sample of urine even from an infant of a few months, while in the case of the child it is as easy every whit as in the case of the adult. In examining children whose symptoms were at all obscure I have for several years insisted on a specimen of the urine being handed to me for examination. I am not content with a mere superficial glance, but submit the sample to as careful chemical examination as I would were the patient an adult. If this is negative, then I go on to make a microscopic investigation after taking the precaution to centrifuge some of the urine. By this means I have been enabled to detect the presence of unsuspected urinary disease, and have repeatedly found crystals, pus, blood, and albumin present when none of these were ever dreamed of.

These remarks on the importance of making a routine examination of the urine in pædiatric practice have a special bearing on cases of pyelitis, many of which are bound to be overlooked if such

an examination is neglected. Bacilluria in the infant and young child is a condition of by no means rare occurrence, and I am convinced that it would be even more frequently discovered if the precaution were taken to examine the urine. Reference to a case which came under my observation a couple of years ago will illustrate this point.

The patient was a female infant of sixteen months who presented a complication of symptoms, including drowsiness, rapid respiration, a quick, feeble pulse, a temperature of 103° F., and marked constipation. There was, in addition, considerable cough, of a short, hacking character. At first the case had been diagnosed as one of pneumonia of the apical type, then the diagnosis had been altered to one of meningitis. *The urine had never been examined.* When I saw the child I was struck by the facts that the pulse, though very feeble, was perfectly regular; that the respirations, though rapid, were not labored. These facts made me doubt the presence of either a pneumonia or a meningitis. On physical examination nothing beyond a few moist sounds was detected, and these were all heard towards the bases of the lungs. I noticed, also, that the face was very slightly puffy. I insisted on examining the urine before giving any opinion on the case. Accordingly a sample was procured. I found it loaded with pus which contained innumerable colon bacilli and a certain amount of albumin, but no blood or casts (the latter were excluded from a centrifuged specimen). I therefore suggested that the case might be one of pyelitis, and advised the administration of 0.2 gramme of *potassii citras* every two hours, and at the same time magnesia to combat the constipation. Under this treatment the case cleared up, the urine gradually became normal, and the little patient (who had by this time been practically given up) made an excellent recovery. It is noteworthy that there was no history of rigors or of urinary symptoms in this case. The puffiness of the face is of some interest, as I have noted this symptom quite recently in another child who presented well-marked evidences of pyelitis.

PUERPERAL PYELITIS

I adopt the term "puerperal" here merely as a matter of convenience, for under this type I would include those cases which occur during pregnancy as well as in the puerperium. It would appear that this form of pyelitis is fairly common, although little or no mention of it is made in text-books. In fact, the literature of the subject seems to be comparatively scanty, and yet it is a condition of very great importance. The exact determining factor of pyelitis occurring during the puerperium is not quite certain. In some cases, doubtless, it is due to infection of the kidney pelvis, either in a general septicæmic process or by extension of inflammation from the

uterus and adjacent organs. It may also be secondary to a cystitis, which in turn may be set up by the passage of a catheter or, in some cases, by bruising of the urethra during labor, which gives rise to an upward extending inflammation. In my opinion, however, the commonest cause by far is infection from the colon. It is a common experience to find that immediately after labor the patient does not have the bowels evacuated until the third or fourth day, and even then the intestines may only be imperfectly emptied. It is an easy matter for organisms whose growth in the colon is favored by constipation to gain an entrance to the kidney pelvis, and set up there a more or less severe form of inflammation.

The clinical picture presented by the puerperal variety of pyelitis is very typical. The attack is preceded by constipation, and comes on with marked suddenness. It is ushered in by a rigor, the temperature rising to 104° F. or even 105° F. To all appearances the patient looks as if she were having an attack of ague. The teeth chatter, and the bed shakes under her. She complains of feeling sick and shivery. The skin is hot, and presently the patient breaks out into a profuse perspiration, in which the whole body is bathed. The attack generally commences in the evening. Tepid sponging will often reduce the temperature somewhat, and during the night the temperature will usually fall to normal or nearly so. The rigor is associated with a very characteristic pain which may be felt in both lumbar regions, but is most commonly limited to the right side. The pain is aggravated by pressure or even by gentle palpation, and tends to radiate along the ureter of the affected side. It is also very characteristic of this pain that the patient is unable to lie on that side. The pain is at first sharp, but later it becomes dull and aching in character.

There is also pain on micturition, and the patient experiences considerable difficulty in passing water on this account. Micturition is not necessarily frequent, as it is usually said to be in this condition, although the patient may have to pass water during the night. On examination the urine will be found to be straw colored, and appears to the superficial observer perfectly clear. On placing it in a urine jar, however, it will at once be seen to be cloudy, this cloudiness being due to flocculent-looking material which consists mostly of pus-cells. The odor is very, very typical, and is best described as

being fishy; the specific gravity is usually quite normal; the reaction is always highly acid. On allowing the urine to stand for some hours the upper portion will become perfectly clear, but at the bottom of the urine glass there will appear a deposit, which may be quite an inch or two in depth. Microscopic examination of the deposit reveals the presence of pus-corpuscles and epithelial débris. A bacteriological examination of a catheter specimen will usually demonstrate the existence of the *Bacillus coli communis* or of one of the colon bacillus group of micro-organisms.

The disease tends, in the majority of cases, to run a very protracted course, the temperature continuing to rise and fall for several weeks. On the other hand, in some of the slighter cases the temperature falls and the symptoms disappear in the course of a week or ten days. These milder cases probably belong to the attacks of puerperal pyrexia for which no cause can be assigned. At all events, it is very likely that a careful examination of the urine in certain of the cases of so-called unexplained puerperal pyrexia would reveal the existence of a pyelitis. Unfortunately, the majority of practitioners seem to be unaware that there is such a condition as puerperal pyelitis, and the urine is but rarely examined by the medical attendant during the puerperium. It would be a wise plan, therefore, to examine the urine carefully whenever there exists a condition of so-called unexplained pyrexia in a puerperal patient.

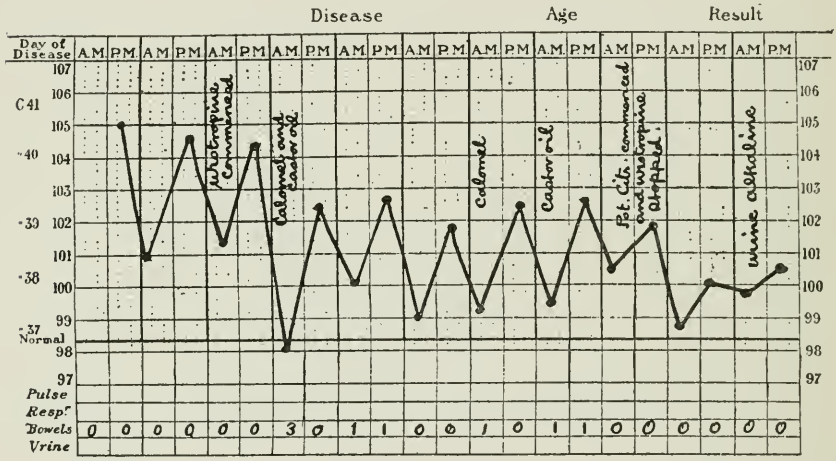
The following report of an actual case will show the importance of keeping in mind the possibility of pyelitis coming on during the puerperium:

The patient was a *primipara*. The labor was somewhat prolonged, as the presentation was an R.O.P. She was under chloroform for three hours, and was delivered by forceps. The perineum had to be stitched, but the wound healed well, and there was no evidence of sepsis. The patient was greatly troubled by constipation. In fact, the bowels did not move until the fifth day, and then only imperfectly. During the first fortnight they only moved three times, and on only one of these occasions was the evacuation at all satisfactory. The patient suffered a good deal from rheumatism, which was doubtless due to the wetting and exposure to which she was subjected by the nurse in cleansing the parts. She was a very rheumatic subject, and hence was readily influenced by damp—a fact which, unfortunately, did not seem to be appreciated by the nurse.

On the seventeenth day after the birth of the child the patient was to have gotten out of bed for the first time, but as she complained of a sharp pain in her right lumbar region, which was thought at the time to be muscular, it was deemed better for her to remain in bed. Suddenly, at 4 P.M., she complained

of feeling queer, and presently she began to shiver. She also had a sharp pain in the back, chiefly toward the right side. The temperature was taken, and found to be 105° F. She was packed round with hot bottles, and extra blankets were put over her. She was also given 1.3 grammes of *sod. salicyl.*, as the condition was supposed to be a rheumatic one, the high temperature at the moment suggesting the onset of rheumatic fever, although the rigor was hardly compatible with this diagnosis. In twenty minutes the patient broke out into a profuse perspiration. The pain lessened, but she complained of extreme thirst. At 5 P.M. the temperature was found to be 104° F. The pain was now quite gone. She was sponged down with tepid water three times, and at 6 P.M. the temperature had fallen to 102.5° F. At 7 P.M. the patient said she felt much easier, and shortly afterwards passed urine. At 9 P.M. the temperature was 99° F. At 9.30 the night-dress was changed, as it was soaked with perspiration. At 1.45

CHART I.



A.M. the temperature was 98.2° F., and at 3.45 A.M. urine was passed. At 5.30 A.M. the thermometer registered 98.4° F. At 8 A.M. urine was again passed. She, however, felt the pain in the back and was very thirsty. On examination the temperature was found to have risen to 101° F. At 8.45 A.M. she felt "cold and shivery." She still had pain at 10 A.M., and the temperature now registered 103° F. In the evening it rose to over 104° F. As the bowels were loaded, an enema was administered, which gave a very poor result. The accompanying chart illustrates the course of the temperature in this case.

On examination the urine was found to contain pus and, naturally, some albumin, but the latter was only slight in amount. The reaction was very acid, and there was a very typical fishy odor which could be noticed whenever one approached the patient's bed. There was always great pain on passing urine, and the patient said micturition caused her to shiver, and her hair felt as if it were standing on end. It was not deemed advisable to take a catheter specimen, but a sample of urine as voided was submitted to bacteriological

examination. The specimen was found to contain an organism belonging to the colon bacillus group.

In this case the patient made a very slow recovery, and became very depressed and melancholic, at times fancying she was going to die. It was fully ten weeks before the pus altogether disappeared from the urine, and even some months afterwards she still complained of occasional pain and difficulty in passing water. At first urotropine was tried, but this was soon abandoned for potassium citrate, under which the symptoms gradually cleared up and the temperature returned to normal.

This brings me to speak more particularly of the treatment of puerperal pyelitis.

It should be a rule in every case of pregnancy and of convalescence after labor to make a routine examination of the urine whenever the patient's temperature is raised. By so doing many cases of pyelitis, even of the milder variety, would be detected which at present, I am sure, exist altogether unnoticed by the physician in charge. I am also inclined to think that no very good purpose is served by keeping the bowels locked up until the third day after confinement. More risk is run by so doing than by having them evacuated, and I might suggest that every puerperal case should have an enema of olive and castor oils on the day after the labor.

When, in spite of every precaution, pyelitis actually sets in, then I have no hesitation in saying that no drug will give such speedy and marked relief as potassium citrate, but it must be given in large doses and at frequent intervals. Two grammes every three hours are not by any means too much to administer. This remedy is somewhat depressing, and may in some cases set up digestive disturbances. These drawbacks, however, are more than compensated for by the gain to the patient in other directions. The drug acts in virtue of its power to change the reaction of the urine, which, under its influence, at first becomes neutral and then markedly alkaline. As soon as an alkaline tide is established the temperature commences to fall, but will rise again if the drug is cut off and the urine once more becomes acid. A curious result of the administration of this drug is that the urine very soon loses its fishy odor, which in all probability is due to the products formed by the presence of the colon bacillus. The latter, it has been stated, will not thrive in an alkaline medium, and hence the importance of rendering the urine strongly alkaline. This explanation, however, I am very much inclined to doubt.

Personally, I do not think urotropine or helmitol nearly so effective in these cases. They are purely urinary antiseptics. Some authorities advise the administration of a combination of one or other of them with potassium citrate, but this is really an incompatibility from which no good result can ever be expected to accrue. The use of an autogenous vaccine may be kept in mind, especially if the symptoms do not improve under the administration of potassium citrate.

INFLUENZAL PYELITIS

This is a condition of which I have had very little personal experience. In such cases, however, the symptoms are usually very severe. The temperature is high and blood, as well as pus and albumin, is often present in the urine. These cases often end in suppuration, so that the condition known as "surgical kidney" is set up. The kidney affected becomes riddled with abscesses, and the only chance of saving life is to have it removed. In this respect influenzal pyelitis pretty much resembles that met with as a complication of enteric fever, diphtheria, scarlet fever, and pyæmia. This is undoubtedly a very severe type and frequently proves fatal in spite of early surgical interference.

TUBERCULOUS PYELITIS

Renal tuberculosis rarely starts in the kidney pelvis. In some instances, however, it does undoubtedly affect the pelvis of the kidney before it attacks the substance of that organ. In all such cases nephritis sooner or later supervenes, and, at all events, the symptoms are rarely typical of a pure pyelitis, but rather resemble those of pyelonephritis of the severe type. In such patients tuberculin sometimes does good, but absolute and prolonged rest and open-air treatment should always be insisted on, and surgical measures early resorted to if the other kidney is found to be healthy.

CALCULOUS PYELITIS

This variety of pyelitis need not be referred to at length, as it is of purely surgical interest, and I am writing from the stand-point of the physician, who is bound to hand over all such cases to the care of the surgeon as soon as the diagnosis has been made.

DEFLORATION PYELITIS

No paper on the subject of pyelitis would probably be deemed complete if reference were not made to this variety which has recently been described by Wildbolz. He maintains that the painful micturition so frequently complained of by newly-married women is often due to pyelitis. In such cases the picture of intestinal catarrh is presented, in others again the symptoms point rather to cystitis, but these symptoms are often followed by pain in the loin, pus in the urine, and a rise of temperature. Wildbolz has recently met with five cases of dysuria in newly-married women. In one case a Gram-positive diplococcus was found to be present, while in the other four the pus showed the presence of the colon bacillus. None of the husbands suffered from urethritis and the gonococcus was absolutely excluded. The pyelitis was on the right side in four out of the five cases. This observer also mentions another case in which pyelitis always followed coitus but completely disappeared so long as sexual intercourse was abstained from. He thinks that the pyelitis of pregnancy, which is relatively common in primiparæ, may really be originally started during the first few sexual acts. I am inclined to think that this so-called "defloration pyelitis" may be set up by the obstinate constipation which unfortunately is frequently a source of trouble to newly-married women who from modesty or some other cause neglect the calls of nature.

In the DIAGNOSIS of pyelitis, besides a careful examination of the urine, pyelography has come to our aid. Our present attainments in this direction are undoubtedly to be credited to the originality of Voelcker, who, we believe, was the first to give us a radiogram of an injected kidney pelvis. To Braasch, however, is due the credit of not only calling attention to Voelcker's work in this direction, but also of continuing this observer's experiments. As the medium of injection into the kidney pelvis through the ureteral catheter both specialists used collargol in 10 or even 15 per cent. solution. This enables the pelvis of the kidney to be clearly outlined and may be used without any fear of harmful effects resulting. By this means a dilated pelvis can readily be made out. Braasch states[1] that in cases of renal tuberculosis "a pyelitis will often show a moderate

[1] *Annals of Surgery*, Nov., 1910.

pelvic dilatation in the collargol plate. On the other hand, with renal tuberculosis a cortical abscess or an irregular pelvis and an inflammatory pyonephrosis may often be outlined and prove of distinct value in differential diagnosis." He has found that such injected radiographs are of considerable practical value in demonstrating the presence of a pyelitis. Undoubtedly, the radiograph is as important in cases of kidney disease as the cystoscope is in diseases of the bladder.

A few additional remarks must be made on the *treatment* of pyelitis. I am firmly convinced that there is only one drug which will control the symptoms in medical cases, and that drug is potassium citrate. It must be given in large doses. Like salicylates in rheumatism, so, too, potassium citrate in pyelitis will not yield good results if given in anything but large amounts. As regards the use of urotropine, I do not think it should be used in pure cases of pyelitis. It is of great service in cystitis, but I have never seen any real benefit follow its administration in cases of urinary disease higher up. Moreover, I am convinced that urotropine is a renal irritant and may actually do harm in such cases. It should, accordingly, be kept for bladder cases, and for these alone, provided the kidneys are found to be healthy. Even when given in such cases, its administration should be intermitted from time to time to prevent the possibility of renal irritation being set up. Boric acid and salol are also excellent in cases of bladder troubles, but they are without value in cases of pure pyelitis. Resinous substances, such as copaiba, are of very little service, as drugs of this class have no effect upon the colon bacillus which is so frequently the cause of the disease.

I would strongly urge the great importance of testing urinary antiseptics clinically, as mere laboratory experiment is of no real value. It is one thing to add some urotropine to a growth of colon bacillus; it is quite a different matter when it is administered to a patient who is suffering from bacillus coli infection.

As regards vaccine treatment, some observers find that this therapeutic measure has a definite effect in ameliorating the symptoms and lowering the temperature even in acute cases. In spite of this fact, however, I am very strongly of opinion that vaccinotherapy ought to be reserved for those chronic cases of pyelitis which have defied treatment with repeated large doses of potassium citrate.

Speaking very generally, such cases do show improvement when a vaccine is employed; the symptoms are ameliorated, and pus and bacilli are reduced considerably, if not entirely removed from the urine. Only autogenous vaccines should be used, and the injections made are every five or six days. It must be remembered that vaccine thereby may fail in cases of mixed infection, such as doubtless exists in many cases of pyelocystitis; but then, again, these are just the cases where potassium citrate in large doses sometimes proves efficacious.

Surgical treatment will be necessary in many cases such as those which result from infectious fevers, the presence of a renal calculus, renal tuberculosis, and such like.

As to diet, milk is excellent, as it acts as a diuretic. Milk puddings, jellies, vegetable soups, minced chicken, toast, tea, and Vichy or soda water should form the principal items in the patient's daily menu. The patient must be kept warm in bed, but it is a good plan to raise the top of the bed or to have the patient propped up on an inclined plane so as to favor drainage of the renal pelvis. I insist on this being done wherever possible. Finally, no case should be regarded as hopeless, and the patient should be encouraged by the medical attendant and nurses, as depression is so often a feature in cases of pyelitis occurring in the adult. Even apparently desperate cases, with continuous high temperature and pus-laden urine, will eventually recover if the condition is diagnosed before the inflammation has had time to spread to the kidney substance, and before the patient is too much exhausted by the high fever and other concomitants of the disease.

SPECIFIC (NEISSERIAN) PROCTORRHŒA

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In a previous dissertation* a vigorous protest was registered against the prevailing erroneous employment of the terms gonorrhœa, blennorhœa, vaginitis, and urethritis instead of the more expressive and exact designations, specific urethrorrhœa, specific vaginourethrorrhœa, etc., to denote genital infection manifestly due to the diplococcus of Neisser. It must be apparent to even the superficial observer that none of the terms mentioned correctly expresses the evident intended meaning; *e.g.,* gonorrhœa literally signifies a flow of semen, blenorrhœa a flow of mucus, urethritis and vaginitis simply indicate inflammation of the anatomic structures from which these names were derived. It would accordingly be equally inexact, inexpressive, and incorrect to speak of gonorrhœa of the rectum or of any other organ or structure implicated in Neisserian infection, hence the superscription selected for this contribution.

Long before, and for several years after, discovery of the diplococcus of Neisser, it was the almost universal impression that, because of its peculiarity of structure and unusual density the rectal mucosa was absolutely immune to Neisserian infection, although the "running issue out of the flesh" has been familiarly known since the beginning of medical history; likewise, that in addition to the urethra the Neisser germ avidiously attacked certain other mucous membranes, particularly the conjunctivæ. It was also formerly believed that Neisserian infection was preëminently a trivial affair and always remained distinctly local, *i.e.,* that the so-called "gonorrhœal toxin" or "gonorrhœal virus" of the older authors never became generally disseminated throughout the human organism. The possibility of vaginal implication was likewise seriously questioned, since it was presumed the normal secretion of the vagina was inimical to the growth and proliferation of the Neisser germ.

* International Clinics, vol. iii, 21st Series, p. 193.

Careful and painstaking clinical, bacteriological, and microscopical investigation, however, demonstrated the inaccuracy and absurdity of the foregoing hypotheses, and the facts are now well recognized that (*a*) the rectal mucosa may be primarily or secondarily attacked by the germ of Neisser; that (*b*) Neisserian infection, instead of remaining distinctly local, may become so widely disseminated as to virtually implicate every organ, structure, and tissue of the human economy and thus constitute a serious menace to life; that (*c*) the vagina is by no means exempt to its obnoxious influence, specific vagino-urethrorrhœa being frequently encountered.

In further emphasis of the statement that Neisserian infection not infrequently becomes widely disseminated throughout the human organism, interpolation in abstract of the views accredited to a few previous writers may be permissible.

Finger[1] admits that many complications and sequelæ of specific urethrorrhœa, such as arthritis, tenosynovitis, bursitis, periostitis, endocarditis, and pleuritis, depend upon the presence of the Neisser germ, which has been carried from the original point of infection.

Two instances of metastatic (?) ophthalmia are cited by Sym,[2] which he differentiates from the form due to direct infection by the fact that both eyes were affected at the same time. He says the condition is severe but not violent; inconsiderable swelling and hyperæmia of the lids and conjunctivæ; slight discharge of thin, mucoid or mucopurulent material containing few or no Neisser germs; ulceration of the cornea is rare and insignificant, and may be due to infection by some other germ. While Neisser germs might be found in the conjunctival and subconjunctival tissues, the author believes this form of ophthalmia is purely toxic.

Eulenburg[3] agrees that arthritis, endocarditis, peritonitis, neuroses, neurasthenia, chorea, sterility, and impotence are diverse possibilities of the Neisser germ. He observed localized lesions resulting therefrom, and differentiated neuralgia, neuritis with paralysis, muscular atrophy with paralysis, and myelitis, citing fourteen instances of neuralgia, four of muscle atrophy with paresis, and one of myelitis. Neuralgia attacked the nervus ischiadicus, n. tibialis, n. radialis, and n. ulnaris; and associated nerves, like the femoral, obturator, and genitocrural, were often implicated. Neuralgia began between two and seven months after the original infection, and muscular

atrophy appeared near the joints affected. One example of myelitis, with endocarditis and arthritis, resembled tabes dorsalis. Cure followed three years of treatment.

McColgan and Cooper [4] saw a woman of forty who had a sudden attack of apparent rheumatism (affecting the thumb and index-finger) whose husband had been under treatment for specific urethrorrhœa, but infection could not be demonstrated in the woman. As no improvement took place in a week, the affected area was incised and a small quantity of purulent material evacuated. The process extended to the hand and necessitated freer exposure later; the flexor tendon became involved, and, after healing took place, was considerably shortened, but no anchylosis was present. The authors considered the condition purely a tendovaginitis. Microscopic examination of the pus revealed Neisser germs.

Columbini [5] describes a case in which the Neisser germs were discovered not only in the urethra and an inguinal abscess, but also in pus from the testis epididymis and parotid gland, and later in the blood and urine. The author claims this is the only case (so far as known) in which an infective process attacked first the testicle and later the parotid gland, although the reverse is common enough.

Campbell [6] mentions diffuse inflammation of the entire upper extremity of one side,* pleuritis, arthritic inflammation, pyæmia, liver enlargement, icterus, peritonitis (in the male), proctitis, felon, etc., due to Neisserian infection. In one case four days after healing of a Pott's fracture pus appeared in the wound and the inflammation continued to extend upward on the thigh, requiring extensive incision for drainage. A culture made from the discharge a month after admission demonstrated the Neisser germs, and it was then discovered the patient suffered from specific urethrorrhœa, which had not

* A somewhat similar case was reported before the American Surgical Association by Powers (*American Medicine*) in 1903. The patient developed an extensive phlegmon about the right elbow-joint with concomitant swelling and œdema from the pectoral region and shoulder to the ends of the fingers. The Neisser germ was found in pus liberated by incision. The course of the disease was protracted over many weeks, the patient recovering with stiffened joints. The author emphasizes the fact that systemic (Neisserian) infection may invade the tissues in wide variety, the germ having been found in the peritoneum, the pleura, the pericardium, myocardium, endocardium, meninges of the brain and spinal cord, periosteum, perichondrium, muscles and tendon sheaths, bursæ, fascia, the skin, the parotid gland, the kidney, and the blood-vessels, as well as the joints.

previously been suspected.* Infection occurred six weeks before the patient was admitted to the hospital. After four months he was discharged with the fracture cured, but there was still a slight urethral discharge. The author states infection by direct contact was impossible in the hospital, and therefore suppuration can only be accounted for in one of two ways: (*a*) that either the Neisser germs circulating in the blood found a point of lowered resistance and there started a suppurative focus, or (*b*) at the time of the accident inoculation may have occurred from germs on the clothing. According to the author, the former is most likely the correct explanation.

Ward [7] believes so-called gonorrhœa is a general toxæmia, the microörganisms that form the toxin being generally localized around a mucous tract. The microörganisms may extend to organs communicating with the infected tract, or penetrate the tissues either by direct extension, as invasion of the peritoneum through the uterus and oviducts, or through the mucous membrane; thence infection may invade the cellular tissues, the lymphatics, the glands, and also the vascular system. Invasion is rendered possible by the paralyzing action of the absorbed toxin upon the leucocytes, which prevents encapsulation of the microörganisms. Having once reached the circulation, the organisms may invade the heart and endocardium, or be carried to the peripheral capillaries, where they may proliferate, produce more toxin, and induce local inflammation. The microorganisms invade the joints, are found in the synovial sacs, in the pleura, pericardium, and are probably present in analogous inflammation of the tendons and periosteum.

Muhlig [8] records a case in which specific urethrorrhœa was complicated by a bilateral infarct of the lungs. He believes there occurred a deferentitis spermatica gonorrhœica (?) which produced a thrombosis of the venæ spermaticæ internæ, whence emboli were transmitted to the lungs.†

* If this statement be true, it would appear that the surgeon and his hospital associates were certainly guilty of gross neglect of duty in not more thoroughly examining the patient and thus ascertaining before the expiration of a month that he suffered from specific urethrorrhœa.

† In the *Münchener med. Wochenschrift* of March 3, 1903, Solomon cites an example of deep ulcers of the labia minora, from the pus of which Neisser germs were isolated in pure culture; and in the March 31, 1903, issue of the same journal Bressel speaks of a man who in the course of an acute attack of specific urethrorrhœa developed violent headache, high fever, pain over the precordia, and on the sixth day definite signs of lobar pneumonia. Blood cultures showed the Neisser germs.

Rugg [9] observed a male of sixty-two during the third day of an attack of specific urethrorrhœa, which responded to astringent and antiseptic injections. Two days later the patient complained of pain in the back; the following day there was great pain in the right wrist, shoulder and knee, and discomfort of less degree upon the left side; later in the day the right wrist became inflamed and swollen, and there was considerable effusion into the right knee, without redness. The left ankle became red and swollen, the left wrist was painful, but there was no swelling. The soles of the feet were painful to touch. The conjunctivæ and sclerotics were injected. The patient was given potassium iodide and opium, and appeared to improve. On the tenth day after appearance of the urethral discharge he became somnolent, then comatose, and finally died, the temperature rising to 107° F. an hour before death.

Hewes [10] mentions nine examples of gonorrhœal (?) rheumatism in three of which Neisser germs were isolated from the blood. The author notes that the victims of specific urethrorrhœa appear unaware of the gravity of the disease, as regards extension by continuity, systemic infection, and transmission to others; and these facts are not sufficiently appreciated by medical men.

Taylor [11] has written an exhaustive dissertation on the rôle of the Neisser germ in disease, and summarizes as follows: (1) It attacks the human urethra, causing catarrhal and hyperplastic inflammation, which may result in chronic arthritis and stricture. (2) It invades the testes and their appendages and thus threatens sterility in men. (3) Though usually limited to the subepithelial connective tissue, it may invade the venous and lymphatic radicles of the male and female genitalia and cause local and general disturbance. (4) The whole organism may thus be attacked, with much resultant suffering and disability. (5) It may cause septicæmia, with serious or fatal result. (6) Arthritis and rheumatism are constant concomitants, causing marked changes in joints, tendons, bursæ, and muscles. (7) After generalized systemic infection the heart may become seriously involved. (8) The gonococcus may be destroyed by the antitoxic influence of the blood-serum. (9) The microbe, or its toxins, may cause serious changes in the cerebrospinal system. (10) Invasion of the pleura may occur, with resultant phlebitis. (11) The spleen may be attacked. (Evidence of liver involvement has not yet been demonstrated.) (12) Many cutaneous

and mucous membrane lesions may be caused by the gonococcus. (13) Bone lesions may result from its virulent action. (14) The entire genito-urinary tract in the male may be involved. (15) The mouth, nares, rectum, umbilicus, and eyes may undergo inflammation from this cause. (16) Infection of the female genitalia may menace life and health. (17) The possible production of an inhibitory serum is undetermined. (18) The gonococcus causes gonorrhœa (?) in the majority of cases. (19) It may be latent, but may also at any time become potential. (20) It is capable of producing the most far-reaching infections to which human beings are susceptible.

In view of the pertinent facts cited, the assertion seems amply warranted that the vast majority of the examples described by the older authors as intractable leucorrhœa (fluor albus), persistent vaginitis, etc., in which cervical, uterine, and tubal involvement almost invariably later occurred, also the persistent and apparently incurable (so-called) urethral and vesical catarrh, originated in infection by the diplococcus of Neisser; and it also appears certain that implication of the rectum, the joints, the peritoneum, as well as other organs and structures which accompanied or followed specific urethral and vaginal infection, were really complications, concomitants, or sequelæ thereof, rather than distinct pathologic entities, as was formerly believed.

Few of the older standard medical text-books even mention the possibility of Neisserian infection of the rectal mucosa, and later authors dismiss the subject with a few lines. While the work of White and Martin [12] accords greater space to description thereof than other books to which the writers have access, yet their consideration of the subject appears markedly inadequate for intelligent and successful guidance, as will be observed by study of the following excerpts:

Gonorrhœal inflammation (Neisserian infection) of the rectal mucous membrane is observed more frequently in women than in men, mainly because women are more exposed to infection from the backward trickling of gonococcus-bearing secretions from the vagina. The disease may also be excited by unnatural practices. The symptoms are those of acute inflammation, viz., free discharge of blood-stained pus, tenesmus, painful defecation, and, on direct examination, acute redness and infiltration of the mucous membrane, with excoria-

tions about the anal orifice. The disease is prone to become chronic, leaving, on subsidence of the general inflammation, one or more localized ulcers which (if allowed to extend) may ultimately cause dense cicatrices. The diagnosis is based upon the presence of the gonococcus, together with a history of infection! The treatment consists (*a*) in relieving the tenesmus and burning pain of the early stages, (*b*) in frequent cleansing of the affected mucous membrane, and (*c*) in applying astringent and antiseptic medications. For the relief of pain and tenesmus, suppositories containing one grain of the watery extract of opium, a quarter grain cocaine, and a quarter grain belladonna will be sufficient! The rectum should be cleansed at least twice daily by hot douches of corrosive sublimate (1 to 20,000) or silver nitrate (1 to 2,000), or, if these solutions occasion severe pain, by a saturated solution of boric acid. When the acute symptoms have subsided, stronger solutions of silver nitrate should be employed (1 to 1,000 and 1 to 500), in smaller quantities. When the general catarrh (?) is cured, leaving only ulcers or hyperæmic patches, these may be touched with a 10 per cent. solution of silver nitrate, or other agents which have proven useful in the treatment of chronic gonorrhœa (specific urethrorrhœa) may be employed. If the discharge persists, a 2 per cent. solution of alum or tannin injected into the rectum will prove beneficial!

An old volume of the *Annual of the Universal Medical Sciences* reviews "gonorrhœa of the rectum" (specific—Neisserian—proctorrhœa) in the following language: Rollet reports one case; Gosselin one; Tardien had never seen one; Boniere experimented and found it exceedingly difficult to inoculate the rectum with gonorrhœal (?) pus, whereas Requin believed it almost sure to follow pederasty with a person suffering from the disease; Kelsey had occasion to suspect its presence but once.* However, in a subsequent volume[13] the

* Even the great Neisser himself has little to say with respect to rectal infection by the germ which bears his name. The following is abstracted from his paper published in the *Medical News* of January 13, 1900: "This affection (specific—Neisserian—proctorrhœa) occurs mostly in women as a consequence of the infection of the rectal mucosa by secretions flowing down from the genitalia. Ulcerative lesions and purulent conditions of the connective tissue around the rectum are sequels which eventually cause stricture, and thus induce permanent invalidism. Sometimes in men and boys, as well as in women, direct infection of the rectum takes place through anal coitus."

subject is treated at greater length: Neuberger (*Russki Vratch*) and Borzeski (*British Gynecological Journal*) observed five cases of rectal gonorrhœa (?) in women. Microscopical examination showed the pus to contain typical Neisser germs when stained by the method of Steinschnider and Galewski. While, in their opinion, ulceration of the rectal mucosa deserves recognition as a pathognomonic sign, they believe Neisserian infection produces only inflammation of the rectal mucosa, ulceration being due to a secondary cause, *i.e.*, mechanical irritation from hardened retained feces, foreign bodies, pederasty, etc. However, the fact still remains there may exist no observable distinctive signs by which these ulcers can be distinguished from those of other origin, *e.g.*, dysenteric, tubercular, traumatic, etc. Histological examination of tissue from one patient afforded no information of value, and the only means of unerring differentiation is microscopic examination of the rectal secretion. Concerning the etiology the authors remark that, so far as is known, (*a*) pederasty is the most frequent cause, (*b*) the spread of pus from the urethra or vagina to the anus, (*c*) the opening of an inflamed Bartholinian gland into the rectum. Prolonged treatment is usually necessary, and even in recent cases the disease may spread widely and recurrence is likely.

Lockwood[14] dscribes an example of gonorrhœal (?) proctitis, endometritis, salpingitis, and oöphoritis which possesses unusual interest because of the following features: (*a*) The specific proctorrhœa attracted attention when no symptoms were apparent of infection of the urethra, the vagina, or the cervix uteri, (*b*) the supervention of endometritis, salpingitis, and ovaritis about a month after appearance of the rectal discharge, (*c*) the efficiency of ichthyol and glycerine tampons in curing the disease without recourse to operative intervention, (*d*) the necessity of searching beyond the vagina for evidences of Neisserian infection, as (according to Bumm) it is rare for the disease to begin in the vagina, usually showing first in the cervix uteri or urethra.

Hartmann[15] records an instance of ano-rectal (Neisserian) blennorrhagia which followed vaginal infection about two weeks; and Lewi[16] refers to specific (Neisserian) proctorrhœa in a child of six years, but fails to state whether or not the diagnosis was verified by microscopical investigation.

Within a period of six months Baer [17] observed in hospital practice nearly two hundred females suffering from specific vaginourethrorrhœa, and in sixty-seven of these the rectum was also involved. He claims infection occurs in three different ways: (*a*) directly through abnormal coitus, (*b*) indirectly from communication with the rectum of some other organ attacked with the disease, and (*c*) by conveyance of germs from without by pus, or by therapeutic measures, thermometers, irrigation, etc.* In the first class the objective symptoms are inconstant, and the statements of patients cannot be implicitly believed. The second mode is rare. The urethral discharge is ordinarily not sufficiently copious to make it responsible, but this is untrue of purulent fluid proceeding from the cervix uteri in metritis. For obvious reasons infection occurs with greater facility in prostitutes and in women who have been pregnant a number of times. The patient usually complains of marked pain on defecation, there is discharge of pus and blood from the anus, the rectal mucosa is congested and bleeds easily in consequence of erosions produced by maceration and epithelial desquamation. Treatment consists in injections of 3 per cent. solution of boric acid, and, to diminish the secretion and destroy the infective agent, solution of nitrate of silver 1 to 3000, employing a quart of each solution. It may be necessary to continue treatment five or six weeks. When an exacerbation occurs, it is difficult to decide whether or not a new infection has taken place.

Murray [19] observed a typical example of primary specific (Neisserian) proctorrhœa in a female, which is abstracted at length, for purpose of illustration: The patient had suffered many years from persistent obstipation, for relief of which she had used various aperients, laxatives, and cathartics; but during the last two years had abandoned these in favor of copious rectal injections of hot water. For several days she had experienced great tenesmus, together with heat, pain, tenderness, and irritation about the anus and rectum,

* Bacon [18] believes that employing for enemata the same syringe previously utilized for vaginal douching is more often than otherwise responsible for infection of the rectal mucosa and the development of specific (Neisserian) proctorrhœa. In the way of treatment he advises that the patient be kept at rest, using a double tube (just within the sphincter) for irrigation purposes, which is gradually inserted, with an outlet for the return fluid, in order to prevent carrying infection higher in the intestinal tract. The rectum should be irrigated several times daily with hot water or mild antiseptic solution.

accompanied by profuse discharge of blood and mucus. There was slight elevation of temperature and increase in frequency of pulse-rate, urine scanty and highly-colored. So nearly did the symptoms simulate those ordinarily due to dysentery that treatment was at first directed toward relief of that disease. The next day all symptoms were increased in severity, there being greater pain with constant desire to urinate and defecate. Frequent copious injections of hot water, starch-water and laudanum were ordered, in addition to rectal suppositories containing opium, iodoform, and belladonna. On the eighth day, the symptoms not having abated, the patient complained of heat and pain about the vulva and vagina with increased vesical tenesmus, which rendered reasonable the suggestion that the disease might be specific (Neisserian infection), and thorough investigation was instituted. The sphincter ani was relaxed and there was some rectal protrusion, the anal mucous membrane being highly inflamed and covered with thick greenish mucopurulent material. A small speculum introduced with difficulty revealed the rectal mucosa intensely red and sensitive, the labia were inflamed, the nymphæ swollen, tender and painful on pressure, although there was no vaginal discharge. Specific (Neisserian) proctorrhœa was suspected, and to confirm this provisional diagnosis cover-glass preparations were made from pus taken from the rectal mucous membrane, and the slides being placed under the microscope were found swarming with Neisser germs. The following day there was considerable vaginal discharge, microscopical examination of which revealed abundant similar diplococci.* The vaginal disease yielded to hot alkaline injections followed by sterilized water, and tamponing with cotton pledgets saturated with solution of silver nitrate, and the rectal infection was treated by saline purgation, copious injections of sodium bicarbonate, boric acid solution, starch-water and laudanum, suppositories of opium, belladonna, sulphocarbolate of zinc, hot poultices and fomentations. The rectal disease ran a protracted course and areas of ulceration supervened, which were subsequently cured by applications of silver nitrate followed by coating with

* To show that this patient was not a pathic, nor her husband a pederast, the author proceeds to explain that the source of infection was a maid suffering from specific vagino-urethrorrhœa who had employed in vaginal irrigations the same syringe afterward utilized by the patient for hot water enemata to relieve constipation.

stearate of zinc and an occasional suppository composed of bismuth and iodoform.

Orton [20] records an example of specific vagino-urethrorrhœa followed by implication of the rectum (also the cervix and uterus). The rectal infection, being secondary, resulted from the vaginal discharge or a syringe previously used for vaginal irrigation. While the patient complained of "pain and an uneasy feeling in the rectum," specific (Neisserian) proctorrhœa was unsuspected until pus was observed to ooze from the anal orifice when the perineum was depressed by the vaginal speculum. The rectal infection was treated first with injections three or four times daily of H_2O_2 one part to water three parts containing sulphocarbolate of zinc five grains to the ounce. Later the patient experienced almost constant desire to defecate, and blood, mucus, and shreds of mucous membrane were passed from the rectum. Treatment was then changed to injections of creolin one-half to three-fourths teaspoonful to a pint and a half of warm water every six to eight hours, alternating with fluidextract of hamamelis four to six drachms to water a pint and a half. This proved efficient, and the rectal complication gradually subsided.

Berndt [21] reports (from Mikulicz's clinic) eighteen cases of rectal stricture due to Neisserian infection, four of the patients being males and fourteen females. According to the author, the stricture arises independently of ulceration, and is the result of chronic infection by the Neisser germ, with inflammatory infiltration of the entire thickness of the rectal wall. The condition was preceded by inflammation of Bartholin's gland in five instances, and in six there was also a history of syphilis. Mikulicz holds, however, that "the majority of cicatricial strictures of the rectum are of gonorrhœal origin, whether the gonococcus be found or not!" In five cases the rectal infection was favored by a relaxed sphincter ani muscle. Stenosis usually occurs one to four inches from the anus, but may extend to the sigmoid flexure. The lumen of the stricture feels tough and scar-like, with a smooth lining, and the rectum is often firmly adherent to surrounding tissues. Hemorrhage is a symptom in nearly seventy-five per cent. of cases, and appears to result from ulceration above the stricture. The treatment consists in (*a*) regulating the bowels, (*b*) in employing antiseptic douches, and (*c*) in dilating the stricture with bougies which should be left *in situ* as

long as circumstances will permit. Division of the stricture by simple posterior incision was tried on three occasions, but was unsuccessful. Radical operation is preferred by the author in the majority of cases, *i.e.,* making a preliminary artificial anus, and then excising the rectum. In two instances the artificial anus was omitted, and one of the patients succumbed promptly to peritonitis. The artificial anus may be later closed if the new rectum satisfactorily performs its function.

In fifty-six cases of specific vulvovaginitis in infants Flugel [22] found twenty per cent. also suffered from specific (Neisserian) proctorrhœa. The subjective symptoms in this class of cases are not marked, and infection usually results from the vaginal pus. The author's treatment consists in suppositories of silver nitrate or ichthyol, and states that the rectal secretion becomes free from Neisser germs before that of the vagina, since the mucous membrane of the rectum affords a less favorable medium for their growth than that of the vagina. He suggests the advisability of searching for rectal infection in all cases of vulvovaginitis infantum.

In discussing primary specific (Neisserian) proctorrhœa in the male, Zobel [23] states careful review of the literature of the previous five years shows little to have been written on the subject, the cases reported having been in women secondary to genital infection; that the disorder in the male is usually the result of pederasty, and may be thus considered primary; that whereas the condition has hitherto been exceedingly rare in this country, since the influx of foreigners from parts of the world in which unnatural practices prevail more examples have been encountered. The three cases described were in American-born males of low intelligence (tramp class), aged sixteen, eighteen, and twenty years, who applied for treatment with the self-made diagnosis of "piles." When informed concerning the nature of the affection, a markedly depressing effect was produced, one of the boys developing a condition resembling the neurasthenia sometimes observed in chronic posterior specific urethrorrhœa. The symptoms briefly summarized were: All complained of anal and rectal soreness on standing, and walking induced greater discomfort. Defecation caused such excruciating pain that they had hesitated to pass feces until constipation had become a serious factor. Two were annoyed by discharge from the anus, and, while the third was

unaware of its presence, a discharge was found on inspection. In one the anal discharge was streaked with blood, and bleeding was noted on defecation. One complained of an itching sensation about an inch above the anal verge, and severe pain was noted on contraction of the sphincter muscle. The patients were feverish, appeared worried and haggard, and felt weak, ill, and distressed. Neither digital nor instrumental investigation was possible at the first visit because of pain occasioned thereby. The author suggests that whenever there is the least suspicion of specific infection about the rectum the patient should be treated as if it actually exists, and the ultimate diagnosis be left for the future; that when treatment has caused subsidence of the acute symptoms investigation will show excoriation and fissures with marked redness and infiltration of the anal and rectal mucous membrane, together with presence of a purulent secretion. Examination of the pus reveals the presence of Neisser germs. He believes specific (Neisserian) proctorrhœa in the male is much more common than suspected by the general profession; that many do not know such a condition can exist. The treatment is directed toward (*a*) keeping the parts clean, (*b*) relieving the severe rectal symptoms, (*c*) reducing the inflammatory exudates, (*d*) maintaining the feces soft, (*e*) healing the ulcerations, and (*f*) destroying the infective agent. The author emphasizes the important fact that, while serious complications and sequelæ, such as arthritis, endocarditis, etc., have not been reported following specific (Neisserian) proctorrhœa, the attendant should remember that rectal examination may furnish the correct clue in obscure cases where definite etiological factors are not readily apparent.

While the foregoing does not embrace all the recorded examples of primary and secondary specific (Neisserian) proctorrhœa, it is believed sufficient illustrative evidence is cited to reasonably emphasize the following pertinent suggestions:

1. That the rectal mucosa is by no means immune to infection by the Neisser germ, as was believed by the older authors.

2. That, on the contrary, both primary and secondary Neisserian infection of the rectum may occur in both males and females at any period from infancy to old age.

3. That while Neisserian infection of the rectum in adults of both sexes is often acquired by pederasty, this does not constitute the sole mode of transmission.

4. That particularly in females (other than prostitutes) and in children, rectal infection by the Neisser germ is more often than otherwise purely accidental, (*a*) by direct extension from an infected vagina, or (*b*) from syringes previously used by infected persons.

5. That, like specific urethrorrhœa and specific vagino-urethrorrhœa, rectal infection by the Neisser germ, when inadequately treated, is prone to become chronic, and may be followed by stenosis as well as other dangerous complications and sequelæ.

6. That, on the other hand, properly and intelligently treated before the supervention of serious mixed infection specific (Neisserian) proctorrhœa is practically as readily curable as urethral and vaginal infection from similar cause.

7. That treatment to be effective must be promptly instituted and be prosecuted with equal thoroughness and persistence as that applicable to Neisserian infection in other situations.

8. That the majority of standard medical text-books are markedly deficient in outlining the management and treatment of specific (Neisserian) proctorrhœa, some of them not even admitting the premise that the rectal mucosa is susceptible to infection by the Neisser germ.

REFERENCES

[1] FINGER: *Wiener med. Wochenschrift*, 1897.
[2] SYM: *Edinburgh Medical Journal*, August, 1904.
[3] EULENBURG: *Medical News*, November 10, 1900.
[4] MCCOLGAN AND COOPER: *Journal of the A. M. A.*, April 2, 1904.
[5] COLOMBINI: *Centralblatt für Bakteriologie*, etc., Dec. 30, 1898.
[6] CAMPBELL: *New York Medical Journal*, January 22, 1908.
[7] WARD: *British Medical Journal*, March 30, 1901.
[8] MÜHLIG: *Münchener med. Wochenschrift*, Dec. 17, 1907.
[9] RUGG: *Lancet (London)*, No. 3605, 1892.
[10] HEWES: *Boston Medical and Surgical Journal*, No. 121, 1894.
[11] TAYLOR: *American Journal of Obstetrics*, January, 1908.
[12] WHITE AND MARTIN: *Genito-Urinary Dis. and Syph.*, 1898.
[13] *Annual Universal Medical Sciences*, v, iii, 1896.
[14] LOCKWOOD: *New York Medical Journal*, 1894.
[15] HARTMANN: *Annals d. Gynec. et d'obstet.*, 1895.
[16] LEWI: *Archives of Pediatrics*, 1895.
[17] BAER: *Deutsche med. Wochenschrift*, 1896.
[18] BACON: *American Gynec. and Obstet. Journal*, Nov., 1898.
[19] MURRAY: *Medical News*, 1896.
[20] ORTON: *Medical News*, 1896.
[21] BERNDT: *Edinburgh Medical Journal*, 1899.
[22] FLUGEL: *Berliner klin. Wochenschrift*, 1905.
[23] ZOBEL: *Medical Fortnightly*, 1909.

DULCAMARA POISONING

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Poisoning by the use of *Dulcamara* berries is of so rare occurrence and so little is written concerning its physiological action upon the human body that the following case should prove interesting to the medical profession. The vine is rather common in its growth, being found as an ornament to many verandas, and it is an odd coincident that so few children have been poisoned by eating the berries, which when ripe hang in such beautiful, tempting clusters. A short description of the plant and its allied species before treating of the case and drawing conclusions may be interesting to many.

Dulcamara solanum, commonly called the bittersweet, belongs to the natural order of the *Solanaceæ.* Other members of this order include belladonna, capsicum, hyoscyamus, *Solanum nigrum,* and tabacum. There are several varieties of this plant, namely:

1. *Solanum nigrum,* the common garden or black nightshade, and containing black berries about the size of peas.
2. *Solanum tubersum,* or the common potato.
3. *Solanum lycopersicum,* or the ordinary tomato.
4. *Solanum pseudocapsicum,* or Jerusalem cherry.
5. *Solanum bacciferum,* or the susumber berry.
6. *Solanum paniculatum,* called Jerubeba in Brazil, is indigenous to South America.

The *Solanum dulcamara,* the bittersweet or woody nightshade, is a climbing shrub with a slender, roundish branching woody stem, which, in favorable situations, rises six or eight feet in height. The leaves are alternate, petiolate, ovate, pointed, veined, soft, smooth, and of a dull-green color. Many, near the top of the stem, are furnished with lateral projections at their base, giving to them a hastate form. Some have the projection only on one side. Most of them are quite entire, some cordate at the base. The flowers are disposed in elegant clusters somewhat analogous to cymes, and standing opposite to the leaves. The calyx is very small, purplish, and divided into five

blunt, persistent segments. The corolla is wheel-shaped, with five-pointed, reflected segments which are of a violet-blue color, with a darker, purple vein running longitudinally through their centre, and two shining greenish spots at the base of each. The filaments are very short, and support large, erect, lemon-yellow anthers, which cohere in the form of a cone around the style. The berries are of an oval shape and a bright scarlet color, and continue to hang in beautiful bunches after the leaves have fallen. This plant is common to Europe and North America. In the United States it extends from New England to Ohio, and is in bloom from June to August. The root and stalk have medicinal properties, though the latter only is official. Bittersweet should be gathered in autumn after the fall of the leaves, and the extreme twigs should be selected. That grown in high and dry situations is said to be the best. The principal alkaloid is solanine. The slight odor and taste, bitter afterwards sweet, is supposed to depend, at least in part, upon this substance. The symptoms produced by this alkaloid are spasmodic labored respiration, dilatation of the pupils, intense dyspnœa, convulsions, paraplegia.

Dulcamara possesses feeble narcotic properties, with the power of increasing the secretions, particularly those of the kidneys and the skin. Dr. George B. Wood observed when the system was under its influence a dark-purplish color of the face and hands and at the same time considerable languor of the circulation. In overdoses it produces nausea, vomiting, faintness, vertigo, and convulsive muscular movements. With this preliminary description, the following history will be of much more interest.

At one o'clock in the afternoon of August 8, 1912, Dr. Gangloff was called to see Ruth W., aged 3. The only symptom of which she complained was cramps in the abdomen, slight tenderness which was general, no fever. Husband's magnesia and one-minim doses of aconite tincture were prescribed. The child was seen again at eight o'clock of the same day. The bowels had moved and were of a normal yellow color and loose. The temperature at this time was 101° F., and the cramps were not relieved. The treatment was continued as stated. At 2 A.M., August 9, convulsions came on, and were so severe as to produce complete opisthotonos. Soapsuds enema, hot mustard tub bath, ice-cap to head, and friction to the body were all tried, with

no apparent effect. The patient was then chloroformed to complete anæsthesia, when relaxation occurred. The child was allowed to come out from the influence of the anæsthesia, when the opisthotonos returned immediately. The same procedure was again instituted and relaxation produced, but with the return of the opisthotonos when the anæsthesia was discontinued. This condition lasted for about two hours, at which time the child lay in an apparently unconscious condition. At intervals of about twenty minutes convulsive muscular movements would occur, and consisted of stretching of the extremities in the most languid manner, the thumbs and forefingers being in extension, the rest of the fingers flexed. These movements lasted from one to two minutes, and passed off with gaping. Any irritation, light, or noise seemed to have a tendency to make them occur more frequently. Strong stimulation to skin would produce a weak cry, but no actual return to consciousness. Sodium bromide had no effect on these movements. The bowels are now moving freely, with paralysis of both the anal and vesical sphincters, and paraplegia. The urine was passing freely in increased quantities and frequency of micturition, with no evidence of acute nephritis. The tongue was partially paralyzed, there being no effort at swallowing except as liquids were placed on the base of that organ, and even then were very feeble in character. At two in the afternoon of the 9th there began to pass in the stools some dark-red, partially-digested berries, which upon examination and inquiry were found to have been eaten from a dulcamara vine growing at the front veranda. Belladonna tincture was then used, but did not affect the muscular movements, which continued to come on at intervals of about twenty minutes. At 9 P.M., in consultation with Dr. Ley, it was decided to push the belladonna and also use chloral hydrate in 3-grain doses.

The patient at this time showed a subnormal temperature, pulse 190 per minute, respirations 40 per minute. Pupils equal, slightly dilated, and reacting to light fairly rapid. Tongue still partially paralyzed, also lower limbs, bowels moving freely, anus wide open, micturition very slight. During the convulsive movements the knee-jerks were exaggerated, partial ankle clonus, and Babinski sign present. When the convulsive movements subsided the knee-jerks were lost, also the ankle clonus, but the Babinski sign remained throughout. Pain could be elicited at all times in any portion of the

body, and caused a weak cry, though no actual consciousness. August 10, at eleven in the morning, the child was in much the same condition of shock. Temperature 96° F., pulse 160, respiration 40. Convulsive movements not so severe. Chloral and belladonna continued. Babinski present, and anal sphincter contracted. Towards evening temperature rose to 103° F. Child died at eight o'clock without regaining consciousness.

An autopsy was performed and showed the following conditions: Pallor of face and body. Blood depending to lower portions of body. No skin rash or marks of violence. Joints normal. Complete dilatation of pupils, eyeballs normal. Abdomen slightly distended with free gas. On opening the abdominal cavity the peritoneum was found to be clear, with no signs of inflammation. Organs all in normal position. Stomach and intestines distended with gas and containing a small amount of dark liquid, which proved to be principally bile. Ascending and transverse colon distended with gas, descending colon and rectum collapsed and empty. Mesenteric vessels more prominent than normal and showing signs of beginning inflammation. The mucous membrane of the intestines, colon, and rectum showed marked inflammatory reaction, with catarrhal discharge also of yellow pus. There were no ulcerations of, nor concretions in, the stomach, intestines, colon, or rectum.

The urinary bladder was about one-third filled with urine.

Liver normal in consistency and appearance. Gall-bladder about one-half filled with bile. Spleen normal. Kidneys normal in consistency. Left, larger than right and composed of several lobes. Thymus gland normal. Pleural cavities normal. Hypostatic congestion in both lungs. Pericardium normal. Heart normal. Small postmortem clot in left ventricle.

Permission to open the cranium and spinal column was not obtained.

In conclusion, the symptoms, in brief, as presented in this case, are: in three to four hours severe abdominal cramps, in six hours fever, in twelve to sixteen hours unconsciousness and convulsions, tetanic in character, producing opisthotonos, which lasted for two hours; this being followed by convulsive muscular movements of an intermittent type and languid in nature, with gaping. Partial paralysis of tongue and lower extremities; also of both sphincters, which

regained their tone before death. Shock and death in sixty hours. It will be noted that these symptoms resemble very closely the physiological action of solanine, of which these berries, though almost ripe, must have contained a considerable percentage. *Solanum dulcamara* is supposed to contain this alkaloid in less quantities than any other variety of this plant.

At first hydruria, later scanty flow of urine.

Paralysis of both sphincters, which regained their tone in about twenty-four hours.

The presence of the Babinski sign at all times in both feet tends to show that there was irritation along the motor tracts or in the motor area of the cortex of the brain. The condition of these tissues would, no doubt, have been found abnormal had they been examined.

The injection of the blood-vessels of the mesentery and the production of the catarrhal and, at some parts of the intestines and colon, of purulent discharge shows a special action of the drug on these tissues.

Medicine

GASTRIC DYSPEPSIA DUE TO INTESTINAL DISEASES AND INTESTINAL INDIGESTION OF GASTRIC ORIGIN*

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In this paper I propose to discuss certain diseases of the stomach and intestines with symptoms referred to other parts of the digestive apparatus than that involved in the pathological process, and as examples I should like to present, on the one hand, a series of cases of achylia gastrica, *i.e.*, complete absence of gastric juice, where the symptoms were entirely intestinal, the so-called gastrogenous diarrhœa, and, on the other, a group of cases, first of duodenal ulcer, and second of chronic appendicitis, where all or the major portion of the symptoms were those of gastric dyspepsia.

The typical picture of gastric dyspepsia, I take it, presents symptoms of a functional disturbance of gastric digestion due to a derangement of its centrifugal and centripetal nervous impulses, the former involving the sensory, the latter the motor and secretory spheres; while in one case the derangement of motor function may dominate the picture, in another case the secretory, in a third the sensory, it is rare that a careful analysis of the case does not show that the triad is affected, although, of course, each in varying degree, and it is only by realizing that these three factors are involved in each case that we arrive at a true conception of the disease.

We must remember that groups of nerves, stimulating or inhibitory, may be cut out by purely nervous influences, calling forth, on the one hand, increased inhibition, on the other increased stimulation, and upsetting for the time being the delicate balance between vagus

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and sympathetic; that these effects may be produced reflexly from diseased conditions elsewhere, preferably from some organ supplied by the same nerves, or that it may be due to the action of substances carried by the blood stream acting directly on the glandular apparatus, on the extra- or intragastric and intestinal nerves, or on the vagus centre in the medulla. Thus many divide dyspepsias into hyper- and hypo-sthenic types, dependent upon whether we have excitation or depression of the neuro-muscular-glandular apparatus.

Of especial interest in this connection are those decomposition products of certain of the amide bases which seem to have a selective action upon the vagus, and which unquestionably play a part in the production of gastric symptoms in certain intestinal diseases, notably those associated with constipation and stasis of the intestinal contents. Thus, in one group of cases the referred symptoms are of reflex nervous origin, in another they are due to circulating toxins, in a third to variations from the normal in the chemical and mechanical properties of the foodstuffs and of the gastric and intestinal secretions.

Realizing this correlation between stomach and intestine, based partly on anatomical relationship, partly on the closely related nerve supply, let us briefly note the salient points in the histories to be presented, discussing, first, cases of achylia gastrica with only intestinal symptoms; second, those cases of duodenal ulcer or chronic appendicitis with symptoms exclusively gastric.

ACHYLIA GASTRICA

The first case showed as its most marked symptom intestinal indigestion with colicky pains, flatulence, and diarrhœa unrelieved by many courses of treatment extending over many years, and directed always toward the intestinal symptoms alone; the second complained of repeated attacks of intestinal indigestion with little or no diarrhœa; the third of recurring attacks of diarrhœa with headache; the fourth of persistent diarrhœa of three years' duration; the fifth of diarrhœa extending over four years, utterly unaffected by treatment; the sixth of attacks of intestinal colic with alternating constipation and diarrhœa. None of these patients complained of gastric symptoms, although two, on being closely questioned, said that there had been an occasional feeling of fulness in the epigastrium. All had

suffered for many years, and had lost some weight, though in no case was this loss very marked; all had been unfortunate in the result of their treatment.

In a further series of 15 cases of benign gastric anacidity gastric symptoms were met in each case, sometimes, it is true, accompanied by intestinal symptoms as well. In each of these six cases with no gastric symptoms the free hydrochloric acid after the Ewald test-meal was always *nil,* the total acidity rarely exceeded 10 (expressed in terms of decinormal sodium hydroxide solution), with one exception pepsin was absent, and as a rule there was little or no mucus. There were two men and four women in the series, of age varying from 30 to 67. As to the cause, in two a severe nervous shock seemed to initiate the symptoms, in two the complaint dated back to childhood, in one there was a history of long and great indiscretions in diet, in two there was an associated splanchnoptosis of marked extent. In addition to a careful dietary, all the cases were given large doses of dilute hydrochloric acid, in all marked improvement occurred, in most a disappearance of the symptoms with a gain in weight, but in only one the reappearance of free hydrochloric acid in the gastric contents.

These cases furnish examples of three types of non-malignant achylia—those of neurotic origin, those due to congenital absence of secretion, and those due to an atrophic gastritis; a fourth group, cases of reflex origin, must always also be considered. Only in the first group can a *restitutio ad integram* be expected; as a rule, these are not true achylias, but are cases of simple achlorhydria, as pepsin and rennin are still present in the gastric secretion. Eppinger, Enriquez, and others have called especial attention to reflex achylias, and some at least of the achylias met with in tuberculosis, pernicious anæmia, marked splanchnoptosis, carcinoma, and tabes are of this group. In nephritis the achylia so often met with and undoubtedly often responsible for the persistent and intractable diarrhœa is usually due to an atrophic gastritis, in other words represents the last stage of a chronic gastric catarrh. Why some cases of achylia should have as their striking symptom diarrhœa, others not, is a difficult question to answer, but faulty chewing and too rapid eating, defective or deficient teeth, and an improper dietary are all probably contributory

factors in the development of intestinal dyspepsia, diarrhœa, and enterocolitis.

We must remember that the hydrochloric acid of the stomach not only aids in proteid digestion, but plays an important rôle in maintaining the tonic closure of the pylorus, in stimulating pancreatic secretion by converting prosecretine into secretine, and in disinfecting the foodstuffs after they reach the stomach; the absence of these latter functions is the cause of many of the symptoms of achylia gastrica. Absence of hydrochloric acid means an open pylorus and a quickly emptied stomach, ergo the small amount of the gastric contents after the test-meal, although this is partly due to the difficulty of aspirating the unchymified food. The entrance of poorly-mixed, mechanically irritating, undisinfected food into the intestine leads to an irritation of the mucous membrane, increased peristalsis, sometimes a true enterocolitis, diarrhœa with large undigested stools, showing often a deficiency of intestinal and pancreatic ferments, and the presence of undigested connective tissue, of diagnostic importance when the test-meal cannot be obtained, and often of lactic acid bacilli. In the true achylias neither hydrochloric acid, pepsin, nor rennin is present in the gastric contents after a test-meal, nor rennin or propepsin in the urine, and the total acid is almost always less than 10. In certain cases gastric pain, due, as a rule, to the eating of dry, coarse foods, a feeling of pressure in the epigastrium, and loss of appetite are complained of; in other cases—gastrogenous diarrhœas—the symptoms are mainly or entirely intestinal; in others there are no symptoms, illustrating anew the possibility of the vicarious action of the pancreatic and intestinal juices. All cases are helped by the care of the teeth, careful chewing, and a bland, non-irritating dietary. In some hydrochloric acid acts magically; in others, especially the congenital type, the gastric mucosa seems to possess a marked hypersensitiveness toward this acid, and it is difficult or impossible to administer it successfully.

The question may well be asked, How can gastric carcinoma be differentiated from a benign achylia? As a rule, the long history, absence of tumor and of obstructive phenomena, increase of motor function, the failure to find occult blood in the stool, the less loss of weight, and the absence of the special tests for carcinoma, those

of Salkowski, Salomon, Wolff, Fisher, etc., aid greatly at reaching a proper conclusion as to the real nature of the disease.

DUODENAL ULCER

When we discuss the reverse of the foregoing picture, that is, the gastric symptoms in intestinal diseases, it is well to remember that the more carefully the so-called functional disturbances of the stomach are studied the more frequently do we find that they are associated with organic diseases elsewhere, notably gall-stones, duodenal ulcer, and chronic appendicitis.

Let us mention briefly the important points in a few cases of duodenal ulcer.

Case 1 complained of attacks of belching, colicky pains four hours after meals and at night, vomiting and epigastric pain, the attacks coming on every one to three weeks. The fasting stomach contained 60 c.c. with no free hydrochloric acid, while after the test-meal 150 c.c. were aspirated with free HCl 19, total acidity 39. The guaiac test showed occult blood in the stool.

Case 2 gave a history of burning pain in the epigastrium, with belching, nausea, cold hands and feet, the symptoms reaching their maximum intensity three to four hours after the heavy meal of the day. The fasting stomach contained 10 c.c. of fluid with free HCl, while after the test-meal 60 c.c. were aspirated with free HCl 9, total acidity 38. The stool showed occult blood.

Case 3 gave a history of attacks of nausea and vomiting, with great depression, vertigo, and pain at 2 o'clock in the night. The stool contained occult blood, the fasting stomach 20 c.c. of fluid with free HCl 11, total acidity 22. while after the test-meal there was an increased amount of juice of normal acidity.

Case 4 complained of attacks of nausea, sometimes associated with vomiting two hours after meals, marked depression, rapid pulse, cold hands and feet, with a great deal of acid fluid in the fasting stomach, and more than 300 c.c. after the test-meal with free HCl 10, total acidity 45, and occult blood in the stool.

Case 5 complained of attacks of nausea, lack of appetite, and pain in the left hypochondrium, the symptoms reaching their maximum about three hours after meals, being relieved by food. The patient had had similar attacks off and on for six years. There were 20 c.c. of fluid in the fasting stomach with free HCl 35, and 150 c.c. after the test-meal with free HCl 65, total acidity 85. The stool showed no occult blood, but the X-ray examination showed a definite obstruction, probably due to adhesions in the upper portion of the duodenum, thus calling attention to the fact that secondary mechanical obstruction may play a part in the production of the symptom-complex of a duodenal ulcer, and probably does so in a considerable proportion of the cases.

Some of these cases showed a zone of hyperæsthesia in the right scapular line at the level of the ninth to the eleventh dorsal vertebra, some in the right epigastrium.

These cases bring out fairly well the salient points of the disease, the true recognition of which has taken place only within the past few years, a disease characterized by the periodicity of the attacks, in the interim the patient, as a rule, being practically well, with pain two to four hours after meals, often increased by lying on the right side, but not so constant a symptom as the pain of gastric ulcer, hunger pain relieved by food, sometimes the disappearance of symptoms after a day's fast, gastric hypersecretion either continual or alimentary and often the microscopic retention of food, in some cases hyperæsthetic zones in the right back and right epigastrium, and often occult blood in the stool, but none in the gastric contents. According to Moynihan, true hunger pain is absolutely diagnostic of duodenal ulcer, while Robert Hutchison believes that continuous hypersecretion is almost always associated with a benign pyloric stenosis or spasm, which in turn is most frequently due to an ulcer in the neighborhood. The French lay especial stress upon this "syndrone pylorique" as a sign of duodenal ulcer. Sometimes we have a mild icterus due to swelling of the papilla of Vater, sometimes an enlarged liver and spleen due to a perihepatitis and perisplenitis, possibly caused by bacterial invasion through the ulcerated surface. The position of the ulcer is usually above the mouth of the pancreatic duct, that is, in a position where the acid chyme can act freely upon the mucous membrane. As to whether secretory neurosis is primary and duodenal ulcer secondary or the reverse it is impossible to say definitely, but the latter seems the more probable.

The ulcer may heal spontaneously or under treatment (rest and a Leube, Lenhartz, or other dietary suitable to gastric ulcer being the treatment employed), or it may lead to a local peritonitis or a contracted scar, it may perforate, or it may be operated upon. The tendency to spontaneous healing is shown in a series of autopsies at Kiel, where there were 11 cases of perforation and 52 cases of spontaneous healing without medical or surgical treatment. While a typical case should be easily recognized, cases with atypical symptoms are most difficult to diagnose correctly and are usually regarded and treated as pure cases of nervous dyspepsia, this error in diagnosis

being helped by the marked nervous, depressive and vagotonic symptoms so often met with in duodenal ulcer. The correct diagnosis is often possible only by the correlation of many little signs and symptoms, each insignificant in itself.

CHRONIC APPENDICITIS

When we consider the close nervous relationship through vagus and sympathetic between the various abdominal viscera, and also in these very organs the chance of toxin formation, notably of the vagotonic amide bases derived from lysin, arginin, etc., and the possibility of their dissemination by the blood current, especially if the antitoxic properties of the liver are in any way impaired, it is not strange that we meet with many intestinal conditions where the symptoms in part or in whole are referred elsewhere, notably to the stomach. Pancreatitis, cholelithiasis, nephritis, and pyelitis may be completely masked as regards symptomatology under the typical picture of a functional disturbance of the stomach, gastric dyspepsia. While nothing is easier to diagnose, as a rule, than acute appendicitis in the adult, nothing is more difficult than certain cases of the chronic form of the disease, except, perhaps, acute appendicitis in very young children, and it is only by a realization of the referred symptoms of the condition produced partly by the constant, although perhaps subconscious, irritation of the appendicular nerves, acting reflexly not only on other branches of the vagus but on the entire nervous system, partly by the increased toxin-formation, probably slight but continuous due to the associated sluggishness of movement in the ileocæcal region, and by a most careful clinical study of every feature of the case, that the proper diagnosis is possible.

Let us mention very briefly the important points in the histories of a few cases in this connection.

Case 1 had suffered from indigestion from childhood, although the attacks were not severe until three years before being seen by us, since when there had been a number of attacks of nausea, epigastric pain, precordial pressure and palpitation, eructation of gas with some constipation, but no pain except in the stomach region. The patient had always been regarded as a psychoneurasthenic, with nervous dyspepsia, and had taken three rest cures with increase of weight but no decrease of symptoms. She had noted that the attacks were more likely to follow exercise or exertion, but when first seen by us nothing was to be made out except a very definite psychoneurosis with

a marked splanchnoptosis; there was no pain on pressure in the appendix region, no increased resistance, no muscle spasm. With the exacerbation of the indigestion, however, although symptoms and complaints were quite typical of a nervous dyspepsia, yet it was found by taking the temperature frequently—and this we believe is of great importance in these cases—that she had occasional slight elevations, the maximum being 99.7°, while four leucocyte counts gave 12,900, 11,100, 10,400, and 10,500 per cm. respectively, with 75 to 80 per cent. of polymorphonuclear neutrophiles. There was also possibly a slight increase in the muscular resistance, a moderate "défense musculaire" over the appendix. The increase of temperature and the leucocytosis, slight though they were, seemed definite enough to suggest a mild inflammatory process, and with the slightly increased resistance in the appendix region an appendicitis larvata in the stage of a mild exacerbation was diagnosed. The operation performed by Dr. Finney showed a chronic appendicitis with many adhesions, some fresh, with constrictions, the tip lying near the gall-bladder. There were no gall-stones nor signs of gastric or duodenal ulcer. Although the patient still has some symptoms of a mild atonic dyspepsia associated with a marked gastroptosis, she has had no more attacks of acute indigestion, no more rise of temperature, no leucocytosis.

Case 2 presented a very similar picture, attacks of nausea sometimes with vomiting, epigastric pain and headache, with regular bowels, but with practically constant gastric indigestion. Regarded by some as a psychoneurasthenic with nervous dyspepsia, by others as a case of atypical hyperthyroidism, she had been given many rest cures without success. There was a marked ptosis of all the abdominal viscera verified by X-ray photographs, but with overfeeding, rest after meals, postural treatment, and the wearing of an abdominal support the gastric symptoms continued. Seen during the increase of these symptoms, and although complaining of no feverishness, the mouth temperature reached 99.8°, the leucocytes 19,000, while on being very carefully questioned she admitted a slight pain on stretching out the right leg, slight tenderness and some increased resistance on very deep pressure at McBurney's point, and slight sluggishness in micturition. The patient herself, however, before being specifically questioned in regard to these points complained only of gastric symptoms. At the operation, again performed by Dr. Finney, the appendix was found to be densely adherent with some fresh adhesions, contained a small concretion and pointed toward the urinary bladder. Since the operation, two years ago, there have been no attacks of acute indigestion.

Case 3 gave a history of persistent epigastric pain extending over many years, with nausea, pyrosis, belching, and occasional vomiting, the symptoms being somewhat relieved by eating; the bowels were regular, there had been occasional slight elevations of temperature, which had always been regarded as neurotic in origin. After several rest and dietetic cures in this country and abroad, each accompanied by great increase of weight, the symptoms persisted. The physical examination showed a moderate degree of splanchnoptosis verified by the X-rays, an increase of the free hydrochloric acid in the gastric contents, a mouth temperature ranging from 98.8° to 101°, leucocyte counts of 13,000, 11,800, and 13,000 on three successive days, with 80 per cent., 78 per cent., and 85 per cent., respectively, of neutrophiles. The signs pointed toward some mild inflammatory process, and although there were no local appendical symp-

toms, from previous experience it was regarded as probable that the case was one of chronic appendicitis. The operation showed a long, adherent appendix posterior to the cæcum with many constricting adhesions, the terminal third being completely shut off thereby and containing a large concretion and a small quantity of pus with fecal odor. After the operation all the symptoms disappeared, including the slight fever, and there has been no return during the succeeding two years.

Among our cases of the past five years we have had 15 similar cases, the diagnosis in all being verified by operation, and all presenting practically the same picture as in the three cases presented in detail—a picture extremely confusing and difficult of proper interpretation and yet with certain strikingly similar features in each case.

First.—The associated ptosis of the abdominal viscera demonstrated by X-ray photographs present in most of the cases, and this, we feel convinced, must be a potent factor—mechanical, in all probability—in the production of many cases of chronic appendicitis.

Second.—The presence of elevations of temperature at some time during the day, especially when the symptoms are most marked, always slight and rarely recognized by the patient, usually increased by exercise or exertion, although this, of course, is not peculiar to chronic appendicitis, but simply represents the condition usually met with in any inflammatory process. Ewald and others have insisted upon the greater disproportion between axillary and rectal temperatures, the normal .5° C. being increased to .7° C., 1° C. or even more.

Third.—The presence of a definite, although slight, leucocytosis, rarely more than 13,000, with relative increase of the neutrophiles to 75 to 85 per cent.

Fourth.—The increase in symptoms after exercise, constipation, or indiscretions in diet.

The gastric contents after the test-meal are often normal, sometimes subacid, oftener hyperacid—the disproportion from normal being usually slight.

Other points may be of service in making the diagnosis—as slight sluggishness to the flow of bismuth in the ileocæcal region, although the bowels, according to the patient, are normal in many cases; the "tache appendiculaire" of the French, the white vertical line on either side of the mouth, with red, rather hectic, cheeks, Rovsing's sign that deep pressure in the left iliac fossa produces pain in the

appendix region, "coxalgia appendiculaire" or the production of pain by the sharp flexion of the right hip, and occasionally slight pain or sluggishness on urination.

And yet in many cases the complaints are directed entirely to the stomach, or the patients complain simply of not feeling well, being easily fatigued, with an irregular appetite; in other words, the usual complaints of the neurasthenic without localization of symptoms.

We must not forget that upon an unstable nervous apparatus—a type of apparatus, for example, frequently associated with that congenital weakness of body structure, splanchnoptosis the effect of a constant or frequent irritant, even though it be so slight as to be unable, or barely able, to rise into consciousness, may lead to a grave psychoneurasthenic state, and, therefore, in cases regarded as psychoneurasthenia with vague gastric or other digestive symptoms it is essential to make our examination so thorough as to absolutely exclude an organic cause. Such an organic cause may prove to be the physical basis of the psychic and nervous disturbances in one so predisposed.

CONCLUSIONS

We have of necessity presented in a very hurried, incomplete fashion these examples of intestinal indigestion of gastric origin, and of gastric dyspepsia due to intestinal disease, and yet we hope that it has shown, in the first place, the comparative frequency of such cases of transference of symptoms from the seat of the disease, and, in the second place, the paramount importance of determining the underlying pathological process if the patient is to be relieved of his symptoms.

The practical conclusions which I believe we are justified in drawing are as follows:

First.—Chronic diarrhœa, especially in old people or those with defective teeth, is so often due to an achylia gastrica that in such cases a test-meal should always be given, and the stool examined for connective tissue, especially after the Schmidt diet. In the gastric contents the ferments should be tested for as well as the acid to determine whether the achlorhydria is functional or organic, and in all achylias a thorough examination of the entire body should be made to determine whether the condition is primary or secondary to

disease elsewhere. In many of these cases the administration of hydrochloric acid in conjunction with a non-irritating dietary will give brilliant results when opium, bismuth, and other sedatives, astringents, and intestinal antiseptics have signally failed.

Second.—In cases with symptoms of gastric dyspepsia characterized by a certain periodicity where the symptoms reach their maximum several hours after meals, where hunger pains relieved by food are present, it is well to always consider the possibility of a duodenal ulcer, and to examine the gastric contents and the stool for occult blood, its absence in the former and presence in the latter being very significant, especially if associated with gastric hypersecretion.

Third.—In all cases of gastric dyspepsia of long duration, especially if the symptoms show exacerbations from time to time, if the patients present the body form so characteristic of splanchnoptosis, and if no success has been met with by treatment based on the diagnosis of a nervous dyspepsia or psychoneurasthenia, the underlying cause is frequently a latent or chronic appendicitis without local manifestations; in such cases it is wise to take frequent temperature observations at rest and after exertion, and to make absolute and differential leucocyte counts. The persistent action of an irritant, such as a mild inflammatory process in the appendical region, especially, as is often the case, with a slight degree of coprostasis in this region with the probable slight increase in toxin-formation, acting upon the susceptible nervous system so frequently associated with this fragile body form—in which chronic appendicitis is so often, although, of course, not exclusively met—may magnify such an unstable sensorium into the typical picture of a psychoneurasthenia, often with symptoms of gastric dyspepsia as the striking single element in the picture. In such cases it is obvious no relief can be expected until the removal of the underlying cause by surgical interference.

SIMULANTS OF NERVOUS EXHAUSTION *

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Probably one of the commonest of the so-called nervous diseases that come for treatment at the present time is what has been popularly termed nervous breakdown or nervous exhaustion. Patients complain of being unable to do their work, constantly feeling tired, with oversensitiveness to irritations of any kind, of inability to concentrate their thoughts on anything, loss of sleep, disturbances of intestinal functions, loss of appetite, inability to eat certain kinds of food without considerable discomfort or even great distress though they used to be able to eat anything they wanted without dread; of oversensitiveness to changes of temperature, great depression on rainy days, despondency over the slightest business worries, hesitancy in undertaking to do anything though it may be quite trivial, of a fear of making mistakes that often keeps them inactive, of a feeling of collapse that comes over them and makes them feel that they cannot stand or walk, of a dread of sinking down in the street, or of fainting or falling somewhere and hurting themselves.

I have preferred to put most of the symptoms into a single rambling, jumbled sentence because that is the way patients usually describe them. They are not satisfied unless they can pour out to you all their feelings, dreads, anxieties, incapacities, worries, and certain physical results of these symptoms, especially regarding digestion, power of taking exercise, and sleep. The number of symptoms that you will hear from any given patient depends to a great extent on how many physicians they have consulted, for questioning them will cause new feelings, or at least new modes of expressing these feelings, to develop and make them think that every slight symptom must be of paramount interest and above all of

* A lecture delivered before the Third and Fourth Year classes in the course in Psychotherapy at Fordham University School of Medicine.

special diagnostic significance to the physician in order to enable him to understand the full import of their condition.

After you have had some experience in practice, particularly if you are situated in a large city, you will realize that this is one of the commonest of ailments of modern times. It does not always develop to the extent of preventing the patient from following his occupation, but it makes him very uncomfortable, seriously impairs his efficiency, and leaves hanging over him an almost constant dread of a breakdown. The condition is probably responsible for more suffering, among the well-to-do-classes particularly, than any other affection that comes to us for treatment. It is often said to be due to the strenuous life in our cities and to the fact that men and women occupy themselves with too many things, until finally the exhausted nervous system refuses to be commanded or controlled any further, and a nervous breakdown results.

Undoubtedly there is such a thing as true nervous exhaustion, that is, the abuse of vital energies and will-power beyond what can be borne, with a consequent disturbance of nutrition because nerve cells do not get the proper opportunity to recuperate their forces. This must be very rare, however, for nature has many checks by which to prevent such abuse of her power. Tiredness and sleep inevitably put an end to really exhausting work after a time. Besides, if you study the lives of the patients who complain of these symptoms supposed to indicate nervous exhaustion you will very often find that their occupations have never been very exacting in character; on the contrary, they are usually people who though much occupied do not *have* to do anything, and are so situated that they can give up when they care to, and that the demands on them are as a rule merely social. On the other hand, while we are supposed to be a people of the strenuous life, let us not forget that this nervous breakdown and so-called exhaustion are much more frequent now than they were at a time of a ruder civilization, when real suffering, continuous hardship, persistent worry, enduring anxiety, and awful strains upon human vitality were much commoner than they now are. When you consider the awful responsibilities, anxiety, unceasing solicitude, and ever-increasing burden of grief and disappointment of a man like General Lee during our Civil War, and remember that he is but a type of many men who have gone through and are now

going through years of such awful responsibilities, the fussy strenuous life of our days, with its many artificial duties, and its striving after mere money advantage, or political or social preferment, fades into insignificance. Generals of armies in actual war do not suffer from breakdown, nor, as our pension list shows, are their lives shortened by the great responsibilities they have to go through, though we might not be surprised if such men had nervous exhaustion in the true sense of the word.

Most of the nervous exhaustion we see around us, however, has come into quite frivolous lives without any real responsibilities, with no serious results depending on the issue of efforts, and with only petty worries to disturb the nerves. There are, however, some instances which will excite your sympathy, and which simulate very closely true nervous exhaustion, and it is these that I propose to study with you to-day. I would not have you think that they do not deserve our professional attention and our heartiest efforts for their cure, though I feel that these efforts have often been entirely misdirected. If I can help you to understand this, then I shall have simplified a problem that frequently proves among the most bothersome that physicians, young or old, have to deal with. The affection is increasing in frequency. It has become very fashionable now to have nervous exhaustion, and almost any lady will tell you that certain of her friends are suffering from it, and many of them will give you details of their own experience with it, express a fear that they have never quite recovered from it, or that it may assert itself again. It is a very common topic of conversation in fashionable circles and as long as this continues the disease is sure to increase rather than diminish, so that you can readily understand its importance.

Case histories represent the only possible way of getting at a knowledge of the affection, its causes, conditions, and possible modes of cure. While I have spoken of it as occurring more frequently among women than among men I would not have you think of it as practically confined to the former or as occurring only infrequently among the latter. I have cases in my notes of strong, vigorous, healthy men, some in early life, others in middle life, and some at sixty, with the strength and health that so often exists at that age. The first one of the cases that I shall discuss with you belongs to this

last group; its origin, course and eventual relief are typical, to my mind, of what these cases mean.

The patient is a man of about sixty-two, who had been forty years on the police force in New York and who had applied for retirement, and was now living in what would generally be regarded ease on his pension. He had accumulated some money which, well invested in property, gave him a good income; he had not the slightest apparent reason for solicitude of any kind; he had asked for retirement not because he felt the burden of the oncoming years, but because he felt that he had done his work and now had a right to give up, to take things easy, to do as he pleased and give himself a chance to live. While on duty he was prevented from indulging in many a phase of city life because of the stern demands of his occupation. He enjoyed ball games, but his post for years had been down-town and one of the pleasures that he looked forward to had been the opportunity to see as many of the better games, at least, as possible. He liked athletic sports and it had required not a little sacrifice to enjoy only the newspapers' accounts of these, when he would have liked to have seen the actual events themselves. He had no taste for reading, but he liked a good show occasionally and his duties practically precluded him from ever getting to one. There were things about the city that he had practically never seen, which he was sure he would like to see, such as collections in the museums, in the zoological garden, in the botanical garden, etc. Besides, New York City was growing out into the suburbs and he felt sure that if he could study the suburbs a little carefully, he could make profitable investments. He was a bachelor and had no cares and no one in particular to whom to leave his money, but thought it would be nice to leave it to various charitable and educational causes, and he had actually picked out some of the institutions that he hoped to benefit by his legacies. I want you to realize that he was a thoroughly sensible man, though even after sixty years quite unable properly to appreciate what would make an adequate and satisfying occupation of mind as life declined.

For over twenty years he had occupied one of the busiest posts in the city. He had been at a crossing where elevated trains ran in four directions above him, where trolley cars ran in four directions around him and, towards the end of his service, the underground had

added its jumble of noise from below the street level. There was a school not far away, and four times a day school children had to be watched passing through the busy streets. Some of the most important department stores of the city were close to him and nearly every afternoon crowds passed the sidewalks, and at holiday time and at particular seasons the congestion of people was hard to manage.

Traffic regulations were not yet so perfect as they have now become and tact was needed to manage drivers and pedestrians, yet keep all moving. A more trying position for nerves in a man who took, or thought he took, responsibility rather seriously could scarcely be imagined. Friends began to assure him that he would break down, unless he gave up his position, and though he ate well, slept well, and laughed off their fears he had some misgivings as to whether he might not be making a mistake in staying so long in so exacting a post.

After a while those who knew that he was perfectly able to live without any anxiety about money matters, or any necessity for the pay that would come to him through his pension, succeeded in persuading him how foolish it was to continue to work so hard when he might live quietly, in complete enjoyment of life, without having to get up at definite hours and be under the stern rod of discipline day in and day out, with the long hours required by the old two platoon system. No wonder then that after a time he felt that in justice to himself he must apply for his retirement.

He secured it without question and then began to enjoy life. There was not a thing in the world that he had to do. If he wanted to lie abed in the morning he could do so, he could go to a ball game whenever he wanted to, and as his tastes were modest he could take up anything he cared to. It would seem as though his life ought to be ideal, and for three months it was *pretty* happy. He had enjoyed a fine vacation. He went to the museums, studied the development of the city, haunted the parks and the zoo, went to the ball games, studied suburban real estate, and made a small investment in this kind of property. The second three months were not quite so happy. Not being in the air as much as formerly, he lost his appetite and with this came worry about the proper kind of food, loss in weight, sleeplessness and constipation. Besides, it happened that his little investment in real estate was not panning out

well. He had evidently been caught by some sharpers and it would be years before developments would take place sufficient to think of any profit, and probably in the meantime much monetary loss would ensue. Now the idea of this loss began to worry him, though it made very little practical difference to him, as it represented not one-tenth of his savings and his pension would have supported him in his modest way even though he lost all his money. He became obsessed with the dread of losing all he had, and pictured himself as a dependent on charity.

His condition brought him to physicians, some of whom told him at the beginning that he had neurasthenia. This is a nice, long, mouth-filling word which means nervous weakness, but which patients think of as representing a very definite affection; if they were told that they were nervously weak instead of being informed that they are neurasthenic the diagnosis would not be nearly so valuable. He was given nerve tonics and nerve sedatives and told to go out more and to stop worrying. He was sure that the medicines did him no good, though he always felt better after having had a talk with the physician. It is one thing, however, to say stop worrying and quite another thing to do it; his efforts not to worry only emphasized the condition. It proved very hard to go out merely for the sake of being in the air, and occasional rainy days would interrupt this routine, which he found it difficult to resume, until his physician once more had warned him of the necessity for open air.

He went from one doctor to another, getting constantly worse, until finally it was quite pitiable to see him. He even cried out of sympathy with himself, growing more impatient with himself for his lack of self-control. He wanted to know why he couldn't be happy and healthy as he had been. He had nothing in the world to do, he had plenty to live on, he could do anything that he liked, could buy any medicine, could go anywhere. Why couldn't the doctors cure him? He had given up his occupation, thinking that he was going to be happy; he had never been so unhappy in life as during the past few months. His health was broken down, he had no appetite, he did not relish what he ate, he was not sleeping at night, and there was a burden of dread hanging over him. Various physicians had told him that he was suffering from nervous exhaustion, the years of responsibility and care in his position as a busy traffic

guider had exhausted all his vitality. He said he had never noticed this until he had been nearly a year off the force, and asked how was it that it was delayed in coming; was there no way by which he could repair his exhausted vitality? These questions were punctuated by tears, and interjections that showed how seriously he was disturbed.

I found his heart in good condition, his arteries certainly not abnormally thickened for his years, nothing pathological in his urine, muscular and joint systems in good condition, and nothing in his nervous system. He ought to be a healthy specimen of manhood from his physical make-up, yet he was intensely miserable and evidently on the high road to further unfortunate developments. I found that he had nothing at all with which to occupy his mind except the worry about his unfortunate property investment, though there was a prospect of his, or his estate at least, eventually getting his money back. As he had meant only to make money for the benefit of others, I tried to assure him that it ought not to make so much difference even if that benefit were delayed a few years.

I told him at once, however, that unless he got something to do he would surely grow worse; his so-called nervous exhaustion had not developed because of his having done too much during the preceding twenty years, but because there was no outlet for his energies now, and as he had been accustomed for a score of years to use up a large amount of nervous and vital energy every day this was accumulating, and finding no outlet was occupying itself within him. He had to find some channel for his vital energies.

It did not take much explanation to make him to understand the truth of this. He accepted my advice at once and told me on his second visit that there was a school-house not far away from his house and that a new trolley line crossed the avenue close to it and he had volunteered to guide the children past there four times every day. They got to know him and got interested in him, and he in them. After a time, his appetite improved, his sleep was much better, his constipation disappeared, regained weight, and began to be himself, though there were still many misgivings whether anything so simple as this could possibly cure a condition that had given so much discomfort. I actually had to prescribe tonics, lest he might think that he could not get better unless he were actively *cured* of his

affection. After a time he found a position as watchman, though his wages were much less than he would have received as a policeman; he had come to realize that he would have to do something; as a consequence he has come to be almost his old self, and now promises to live at least ten years more, though five years ago he thought that he might as well prepare for the end.

This is a typical case of what it seems to me is frequently called nervous exhaustion. It is not any such thing, but the symptoms are due to short-circuiting, to use the electrical term, of nervous energy within the system. If the energy manufactured by a dynamo does not find work to do outside of the machine, it may be diverted to producing harmful effects within the mechanism. This seems to happen in many people—particularly in those who have formed habits of developing much energy—if they try to do little after having worked hard for a long period of time.

A typical example among woman patients is easier to find, and usually comes in connection with the intense prolonged strain often required by the care of an invalid parent during months or sometimes years when an inevitable fatal termination is approaching. The cases are so common that you will surely see one or more of them within a year or two of your graduation and they are often puzzling and refractory to treatment, though successfully treated they bring you a corresponding meed of praise, for these patients, especially if their case has been misunderstood by others, are very grateful for any relief afforded them and above all for a cure that may come not so much through medicine as through a change in habits of life and a recognition of the real cause of their symptoms.

Perhaps the most interesting case in my notes is that of a young woman who had nursed her mother after a stroke of apoplexy for some two and one-half years. Mother had been an active, vigorous woman up to the age of sixty-five, able to do anything that she wished, and being unaccustomed to invalidism, disinclined to see her friends, because of the paralytic condition, and with fear for the future hanging over her and not much interest in books to relieve the tedium. It is easy to understand how much occasion there was for the development of neurotic symptoms. She required the attention of her daughter every moment of the day and a good deal of the night. Her appetite failed as a consequence of her inactivity, worry, lack of air and occupation of mind, and it was not long before she suffered much

from sleeplessness, and then spells of crying and other hysterical manifestations came on the scene. I know nothing more trying than constant association with such a patient. The daughter filled the post of nurse faithfully and well. She was watched carefully herself to be sure that her health would not suffer and she actually seemed to thrive on her duties. She took advice well, went out regularly, ate at regular hours and as a consequence was able to stand the strain, though for a time it was rather hard to get her to take certain diversions that seemed necessary for her. After the first six months she settled down into the nursing as if trained to it and for two years was a model, devoted daughter and nurse, keeping good control of her patient and generally exhibiting plenty of character.

Her mother's death might almost have been looked upon as a relief rather than a misfortune. The patient was perfectly willing and ready to go and seemed to realize particularly that she had been a burden.

Within a month after the mother's death her daughter seemed to have recovered any loss of buoyancy of disposition that came with the loss of her mother and there seemed to be no after-results of her long and arduous tasks. After about three months, however, some symptoms of nervous irritability were noted, nervous indigestion developed, there was the usual diet tinkering, and then after a time constipation and insomnia appeared. At the end of six months there was a marked picture of neurasthenia with inability to do things, dissatisfaction with life, and many other symptoms of what is called nervous exhaustion. A physician whom she consulted told her that evidently during the long siege of caring for her mother she had exhausted her nervous energies and that for a considerable period, until her exhausted nervous system would recover, or until she would have accumulated a store of new nervous energy, she must expect to have all her functions, and particularly her digestive and intestinal functions, disturbed, her appetite lessened, her power to do things impaired, her ability to think interfered with and her sleep considerably disturbed. The strong influence of a suggestion of that kind from a physician can be readily understood. She settled down to months of incapacity, waiting for her energies to be renewed. After months of waiting, however, with no improvement, but, on the contrary, further deterioration of symptoms, she became anxious and

could not be convinced but that she had so exhausted her nervous energies that she would never be able to take up her work again.

Her brother, a very sensible clergyman, after noting her condition for some time, said to me, when asking me to see her, that he felt sure that the one thing that would cure her would be if she had another invalid to care for. Of course she would have to be as much interested in the case as she was in the previous one, would have to take as much care of it from a sense of duty so as to forget herself, and then all would probably be well. I agreed with him that probably this was the real status of the case. I found that the young woman had no heart affection, no pathological condition of her urine and no disturbance of her nervous system except the subjective symptoms of which she complained and certain objective ones that followed as a result of her over-attention to herself, lack of exercise, disturbance of appetite and sleeplessness.

It is rather important in all of these cases to look carefully to the urine, for sometimes what seems to be a nervous exhaustion or a so-called neurasthenia is the preliminary condition of the systemic manifestation of Bright's disease. In our first class of students here at Fordham one of the young men was somewhat the butt of ridicule on the part of his fellow students because he always complained of so many symptoms. It happened that once during his fourth year he was asked to supply a sample of his urine for the study of the class, with the idea that it would be normal for contrast with pathological specimens. To the surprise of all it was found loaded with albumin. There had never been any previous definite indications of Bright's disease. There had been no œdema and no special kidney signs. Further urinary examinations confirmed the first finding. He was not told of this, but his symptoms now developed rapidly. He died after a series of convulsions some six weeks later, treatment having been of no avail.

After careful determination that there was no organic basis for the symptoms occupation was found for the young woman. It was suggested to her that her conduct as a nurse had shown her ability in this matter and that perhaps she might find a vocation in nursing that would prove eminently suitable and satisfactory for her. She demurred and had been so long nursing the phantom of nervous exhaustion that she could not think of taking up anything seriously

until she had recovered her nervous capacity. After a time, however, she was tempted to care for a distant relative suffering from cancer and found very soon that just as soon as she became interested she began to improve. Training in how to live, definite directions for eating, for she had lost in weight, and for regular outdoor exercise, soon made a great change in her health. How this change could be accomplished otherwise would be indeed difficult to understand.

In order to keep the balance the next case should be a man, and this time it is a clergyman. He had built a church under rather difficult circumstances and had been very much occupied with the responsibilities for the payments on it when due. As he said himself afterwards, no sooner was one note paid than the pay day for another loomed up, and the panic had seriously disturbed his people and his financial arrangements and a strike in the locality had thrown him back until he feared that he might not be able to accomplish his purpose; in spite of discouragement of every kind, however, he had succeeded in meeting his obligations without much difficulty.

Everything turned out very well. The last payment was made and he felt free. He had looked forward to the time when the burden of responsibility would be lifted and felt that he would sleep better, eat better, and could do many things that he had to put aside because he was so much occupied with the building and paying for his church for the past twelve years. But the habit of twelve years is not easy to break. It was hard to find something else to do now in which he would be compelled to be as much interested as he had been in his church building. He began to lose his appetite and sleep. While he formerly awoke with a definite feeling of his responsibility rather stimulating as a rule, now his waking was likely to be associated with an indefinite feeling of something hanging over him that he found much harder to dissipate. He began to have uncomfortable feelings in the region of the stomach after eating, and was sure that he was suffering from indigestion. He removed, particularly, the fats and starches from his diet. He lost weight and it was not long before constipation became marked. He became irritable and preoccupied with himself. His physician told him that this was the result of a nervous breakdown as a consequence of the long years of responsibility with the worry and anxiety that he had gone through. He was advised to take a long rest and went to Europe for three months.

The change worked wonders for him. He regained his appetite and weight and he came back quite sure that he was now himself again. It was not long, however, before some of his old symptoms returned. He had lost his taste for reading to a great extent during his intense occupation with business matters, and while his ordinary duties as a clergyman served as some diversion they were not entirely engrossing. Besides, he had now become an old pastor with three assistants and they made it their business to relieve him of every possible care with the very commendable idea of making his mind easy so that his health might not be disturbed, but with the result that he has comparatively little to occupy him.

I am sorry to say that I have not accomplished as much in this case as I would like to. He is a man of sixty with no organic disturbance, but it seems quite impossible to occupy his mind enough so as to prevent a certain overconsciousness of the sensations and feelings produced by his physiological functions, which he has brought too prominently forward by concentrating attention on them. He has learned, however, that what he needs is diversion of mind rather than correction of ills of his body and that he is using up energies within himself to his own detriment. Unfortunately he became so occupied with one phase of work without sufficient attention to his intellectual life that now that his magnificent church property is paid for he lacks vital interests. He has realized this fact to some degree and he is gradually working up other interests, but he has many setbacks and occasionally relapses into the feelings that he probably exhausted his nervous vitality by overwork during the church building period.

I have seen conditions of almost this same kind develop in business women. The main source of nervous breakdown among women in preceding generations used to be disappointment in love, and grief for relatives who had died, especially when preceded by tedious care and nursing. Since women have gone more into business, however, we find that disappointment in business affairs and business worries and cares produce just the same effect. I recall some years ago a widow whose husband died during a time of financial stress, leaving his affairs very much involved. They had no children and she had been considerable of an invalid. She frequented health resorts, had taken up various health fads, and had turned to

"pathies" of various kinds to be relieved of rather vague neurotic symptoms. When her husband died rather unexpectedly, it would ordinarily be expected that she would go to pieces under the added responsibilities. For a time after she realized the tangled condition of his affairs she was very much depressed, but she was a business man's daughter, and she realized that if the business was not cared for by some responsible person deeply interested in it she would be left almost in want. She took hold and very soon developed an excellent faculty for business details. There were those who said that in managing her husband's uncompleted contracts she proved to have a better business head than he had ever had. This would have been quite undiscovered only for the crisis that compelled it.

At once she began to improve in health, her symptoms disappeared to a great extent even during the first few months, and at the end of a year she confessed that in spite of the occupation which made calls on her for many hours every day she had not been in better health for years. At the end of three years she closed out the business with a comfortable fortune. She looked forward then to years of happiness, having learned the value of life by living it. I am sorry to say that the end of the story, while very interesting, is not nearly so happy as might be expected. She had no social ambition, and had never had any particular interest in literature or music, and now that she was left once more with very little to occupy her, she noticed a return of the old symptoms. At first she shook them off, but after a while, especially after having consulted various physicians who talked neurasthenia and nervous exhaustion to her, she became convinced that during the three years that she had devoted herself to business she had exhausted her nervous vitality and was now suffering from the modern fashionable ill, nervous breakdown or prostration.

After some months she went to Europe and was much benefited but not entirely relieved. When she came back and tried to settle down to the quiet simple life that she thinks eminently suitable for her, her old symptoms began to recur, she lost her appetite and sleep, her digestion was disturbed, etc. I have found it very hard to make her understand that her years of occupation with business, far from representing nervous exhaustion processes, really represented proper employment of her vital energies, with the needed dissipation of the force that accumulates as a consequence of her being a living being.

It has to be used up either externally to her in doing something, or as the modern expression goes it will "*do*" her. When she becomes convinced of that and finds some occupation in club organization, charity, politics, or some other of the many ways that are possible to her, for she feels she cannot go back to business, she will be well again. In the meantime she is inclined to think of herself rather complacently as an invalid, as a consequence of her years of work in settling up her husband's affairs.

As to the treatment of these cases, while the mental state is by far the most important factor of causation and for cure, the physical factors are extremely important. The lack of any occupation of mind that has a gripping interest will almost nullify any other treatment until a change in the mental state is brought about. Not alone occupation but interests must be found for these people. To get them routine work to which they do not bring ever renewed interest as time goes on will be at most a temporary expedient. It is very often a difficult matter to secure such occupation of mind, but that is the real problem in therapeutics. These people go round and are improved by various new fads, medical, dietetic, hypnotic, hydropathic, mechanical, which simply means that for a while their interest is so much taken up with their cure that they forget about themselves. Various health resorts prove beneficial to them because they arouse their interest. Change of air and scene usually do them good, but their symptoms recur when they try to settle down to ordinary life at home. They must simply get interests, and if necessary these must be created for them. Women above all need interests in other people. Charities of various kinds and particularly the care of children—crippled children have the highest appeal—will do them good.

In many of these patients some comparatively slight physical disturbance will be found to be at work. It may seem entirely too trivial to produce any serious ailment and yet successful treatment of it will often give the greatest relief. The reason is that in such people any disturbance of sensation that is constantly present becomes exaggerated and takes on serious pathological significance. Beginning flat-foot will, for instance, in such patients produce tired feelings in the legs which are complained of as almost unbearable aches. Any trouble with the eyes is sure to be the source of many reflexes.

Disturbances of breathing are particularly likely to prove irritating and exhausting to them. If they follow any occupation that requires frequent use of one group of muscles, such as writing, or playing on a musical instrument, ironing, standing or the like, there are sure to be serious complaints of muscular achiness. These local conditions must be carefully treated. They are not the cause of the general condition, but they tend to emphasize it and make it persist. The cure of even these trivial conditions will often mean the beginning of general recovery. That is why magic shoes, glasses, treatment of the nose, and the like are sometimes proclaimed as remedies for nervous exhaustion. The correction of any continuous symptom is an important auxiliary for the treatment of the neurotic condition.

At the beginning of their treatment the physical factors must be cared for. Patients are often sleepless and taking drugs for sleep. These must be dropped, the patients must be sent out into the air,—not to walk, nor get tired, but to get air,—for from eight to twelve hours a day. An ocean voyage is fine and people sleep all day on deck and then all night in their state-rooms. Sleep is often simply a question of air. These patients have often lost in weight, and this must be corrected by an increase in the amount of food. Dictate what you want them to take at breakfast, at luncheon and at dinner, and see that they take it, and then a cup of milk and crackers before going to bed, and in warm weather a dish of ice cream in the afternoon. Sleep will improve, and constipation be relieved as soon as they eat enough. Have them weigh themselves; they will get interested in seeing themselves get heavier and in learning that they can live much more normally than they thought. This interest will be a good diversion of mind for a considerable period. In the meantime if they are to continue well the question of an interest in life must come up. This is the real therapeutic problem for permanent cure and without it there will be loss of interest in your method of treatment after a time, and then they will make their way around to successive doctors to say that they have tried every one and no one can do them any good.

NATURAL SELECTION: THE ETIOLOGICAL FACTOR IN THE DECREASE OF TUBERCULOSIS

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There is no doubt that deaths from tuberculosis are decreasing in most States of the Union, but the general death rate prior to forty years of age is also decreasing, and there are no reliable statistics which indicate that tuberculosis of the lungs as a cause of death is decreasing more rapidly than the general mortality. Prof. Walter F. Willcox, consulting statistician of New York State, in his Report, dated June 21, 1911, says (p. 14): "Neither in the city [New York] nor in the rest of the State has the proportion of deaths due to this cause [tuberculosis] undergone any steady and material reduction during the decade 1900–1909."

The English Registrar-General of Vital Statistics, writing on October 19, 1910 (private letter), said that his "annual report for 1908 does not indicate any very marked change in the rate of mortality from tuberculosis disease since 1884," the date of Koch's discovery of the tubercle bacillus, and Dr. Maurice Fishberg, of New York, is authority for the assertion that in Germany the death rate from tuberculosis has, for the last five years, ceased to fall.

As there is no doubt that deaths due to what appears to have been tuberculosis were decreasing before Koch's discovery, the decrease which has occurred since that event cannot be attributed to efforts to stamp out the disease which have been made during the past quarter of a century—efforts which were not made, and could not have been employed until the cause of the disease had been ascertained.

In the United States two other factors in the "decrease" of tuberculosis must also be taken into consideration. The first is false registration of the cause of death, and it must be admitted that living tuberculosis of the lungs is not systematically reported in those States in which the only law requiring this procedure is a city ordinance. Unreported living tuberculosis naturally becomes

"bronchopneumonia," "chronic bronchitis," or "pneumonia" after death has taken place. The second factor is death of the tubercular patient away from his or her residence. It is certain that the only citizens of Eastern States who remain at their homes after becoming tuberculous are those who have not sufficient money to go to Colorado or California, or elsewhere, and that the mortality statistics of most Eastern States do not show the complete tuberculosis death rate. Upon the other hand, the tuberculosis death rate of Colorado and California is abnormally augmented by the deaths of a great number of non-residents. Nor is this all. Europeans, chiefly residents of New York City, except Jews, who have been counted in United States census, very often return to their native lands as soon as they become sick, often with tuberculosis, but their deaths in Europe never reach our records.

If, however, deaths due to tuberculosis of the lungs were decreasing prior to Koch's discovery, and are still decreasing, some reason, altogether outside man's efforts, which only began in earnest twenty years ago, must certainly exist. The object of this article is to explain what that reason is—in other words, to show what caused deaths from tuberculosis to decrease before human efforts in that direction were able to accomplish anything worthy of the name, and to indicate what natural force is still at work.

Some biologists assert that man's evolution has ceased; the human race, they say, has become civilized, and has obtained so great a mastery over the forces of Nature that stringent selection no longer takes place. Hunger and thirst seldom cause death; wild beasts have ceased to be a serious danger; man's human enemies destroy comparatively few lives. Neither courage nor intelligence is rewarded with long life and many offspring. The weakling and the illiterate raise as many children in the slums as do stronger and wiser men in hygienic homes.

To some extent these authorities may be right, but they ignore the important fact that, although the direction of the evolutionary process may have changed, natural selection has by no means ceased. Indeed, its speed has not slackened. On the contrary, its action to-day is probably as great as it has been at any period in the life-history of man. But many biologists who are occupied chiefly with physical characters or with mental traits have failed to observe

this change of direction. It is, nevertheless, both real and capable of demonstration. If human evolution has ceased, if natural selection no longer eliminates the unfit among civilized peoples, it is certain either that elimination is not selective or that everybody dies of old age or haphazard. The untruth of the second alternative is obvious. As to the first, it is undeniable that, even in the United States, a highly-civilized country, millions of people die before or during the child-producing age, nearly all of whom are eliminated because they are incapable of surviving under the ordinary conditions of the environment in which they find themselves. Moreover, where, as in all civilized communities, men live in dense and settled masses, and so can take disease from one another, a vast number of deaths must be attributed to zymotic disorders. Turning to specific cases, if elimination by disease has ceased to be selective, then every individual who drinks typhoid-infected water must not only contract typhoid fever, but must also die of that malady, and every man, woman, and child who is at any time attacked by tubercle bacilli must invariably die of tuberculosis. That tuberculosis does "select" is proved by the fact that practically all cadavera show signs of having at some period been attacked by tubercle bacilli, but in most cases the attack has not been sufficiently powerful to produce marked disease, and death has occurred, often many years later, from entirely different causes. As is well known, from one-fourth to one-seventh of all deaths in the civilized world are due to tuberculosis, yet if elimination by this disorder has ceased to be selective, *all* deaths in the civilized world must necessarily be caused by it, for practically everybody is attacked by it at some period of existence. In the face of such evidence as this, it seems improbable that any educated man will deny that zymotic disease is a part of the process of natural selection.

Until recent years, acute tuberculosis of the lungs was not an uncommon cause of death, and thirty years ago it was, in large cities, a frequent cause. Although no physician professes to have succeeded in curing a case of acute phthisis, it is to-day a rare condition. Why is this? The tendency toward tuberculosis is a congenital trait—a germinal variation—which is passed on from one generation to another. When both parents and a number of ancestors have possessed the tubercular predisposition, the outward manifestations

of which consist of long, narrow, flat chest, delicate lungs, and feeble resisting power to certain micro-organisms, the offspring generally presents an exceptionally favorable medium for the propagation of the tubercle bacillus, and the victim usually dies from phthisis before he or she has reached the age of twenty-five, and not infrequently at a much earlier age.

Let us emphasize the exact facts. The comparative rarity of deaths from acute phthisis to-day cannot be due to cure of this malignant form of tuberculosis, because physicians do not profess to have cured it, or to be able to cure it. It cannot be the result of prophylaxis in the form of fresh air and a special diet before symptoms of the disease have made their appearance, because very few sufferers have been financially able to obtain such prophylactic treatment, and still fewer have been willing to leave their homes to escape infection when no actual disease existed, although they presented symptoms of special predisposition toward tuberculosis. The infrequency of acute tuberculosis of the lungs cannot be a result of residence in high altitudes, where tubercle bacilli are not numerous, for few sufferers have ever reached such places, and most of those who did reach them ended their days there. The rarity of acute phthisis is the direct result of natural selection—the slow, but gradual, elimination by tuberculosis of the lungs of those individuals who were born with the tubercular tendency in its extreme form before they were old enough to beget children, who would, in all probability, have inherited their congenital susceptibility toward tuberculosis.

Tuberculosis, it should be remembered, differs widely from, for example, scarlet fever or smallpox. While all three diseases must be classed as contagious, the first-named does not seriously injure any person who can correctly be described as being in good general health. The most extreme supporters of the contagion doctrine in reference to tuberculosis admit that when the tendency, predisposition, susceptibility, diathesis (or whatever the condition may be called) is absent, tubercle bacilli never make a sufficiently successful inroad upon the human body to produce tuberculosis.

It is undeniable that persons vary greatly in the susceptibility to infection and in their powers of subsequent resistance. If, then, a lethal disease is very prevalent, it is certain that it presents a stringent form of natural selection. In our country hardly any one

escapes measles, whooping-cough—or tuberculosis, often of such a mild character that he is ignorant of the attack—unless he possesses inborn immunity. Those who escape death from these disorders possess resisting powers toward the microbes which produce them. Whenever any form of selection is stringent, it is accompanied by an evolution of those qualities which enable the survivors to live. It follows in the case of disease that selection will cause an evolution of an inborn power of resisting infection, or of an inborn power of recovering from infection. The two powers are quite distinct, and are sometimes used against quite distinct diseases. Only inborn immunity—inborn power of resisting infection—is of much value against tuberculosis, for it is well recognized that recovery from tuberculosis does not confer immunity, or even increased resisting power.

When a person who has suffered from, and recovered from, a serious attack of tuberculosis possesses increased resisting power, there is always good reason for believing that his added security is due solely to improved conditions of general health and surroundings, increased knowledge of what conditions should be avoided, etc., and never to any physical advantage conferred by the disease. As tuberculosis weeds out those who cannot resist infection, it is evident that, if natural selection acts at all—and no scientific man will deny that it does act—it will cause an evolution of inborn immunity. Tuberculosis always tends to weaken the individual afflicted by it; it is not known to confer any kind of benefit. All races and nations who have been exposed to it are resistant to it precisely in proportion to their past experience of it. For example, Europeans who have suffered severely from tuberculosis for hundreds of years often resist dangerous infection by it, or, when infected, recover from it more frequently than negroes, who have suffered less on account of living outdoors in Africa for many generations, and much more frequently than (American) Indians, who, until late years, had had no experience of it, and who are so susceptible to its attacks that they only survive by living in the open air, or in tents which do not exclude the air. There is no doubt that great numbers of negroes were taken from Africa to Europe as slaves some hundreds of years ago, but to-day no trace of them remains. Indeed, an African negro or one of his descendants is a greater curiosity on

the streets of London or Paris than a native of India is on the streets of New York. The microbes of tuberculosis, which flourish in the crowded cities of the Old World, wiped out the susceptible black men, whose race had not undergone selection by this disease in their native land, because outdoor life had always been their custom, as well as that of other uncivilized peoples. It is well known, of course, that the parasitic tubercle bacillus does not flourish in the open air where sunlight and oxygen are plentiful.

It is at present the fashion to profess to believe that no racial susceptibility toward tuberculosis exists and that what is commonly described as racial predisposition is chiefly the action of the environment. To some extent this idea may be correct, for in many instances the Jews are referred to, and there is, most assuredly, no Jewish race, the term "race" being synonymous with "sub-species" or "variety." This fact, however, is of extremely little importance, because Jews unquestionably have distinctive characteristics, one of which is that, while they are by no means immune to attacks of tubercle bacilli, they are almost immune to death from tuberculosis. This point will be discussed later.

Returning to the case of the colored people. There is no doubt that the death rate among negroes in our country can be reduced by improving their environment, and by inducing them to burn the sputum of all consumptives; but in crowded cities tubercle bacilli are so abundant and have such ample means of propagation that those colored people who are markedly susceptible, as a consequence of their race never having passed through tuberculosis selection, are likely to continue to succumb in great numbers. In a few words, the negro people are for the first time undergoing tuberculosis evolution in the United States to-day, and as yet no human device has materially altered this cosmic procedure.

The Jews have for many generations been an urban people. Self-protection has compelled them to live in congested city districts under most unhygienic conditions. As a result, they have been rigidly selected by the microbes of tuberculosis, and only those have survived who had little congenital tendency toward the "great white plague." In modern times and in a few countries persecution of Jews has ceased, and as a consequence the environment has greatly improved, especially in the United States and Great Britain, where

Jewish persecution is practically unknown. Side by side with the improvement of the surroundings has gone less stringent selection by the tubercle bacillus, which means that some Jews have survived who, under former conditions, would have died in infancy. The change has, of course, been accompanied by a reduction of the Jewish death rate and, in addition, a lowering of resisting power of Jews to tuberculosis in consequence of the survival of individuals who were born with the phthisical diathesis and who lived long enough to beget children who inherited that predisposition. What has been described represents the actual condition in New York City to-day, where phthisis among Jews is far from uncommon, although it seems to cause few Jewish deaths. Although the physique of Jews is very often poor, and their chest expansion meagre, yet they live through tuberculosis pulmonalis, and die of some other malady. Upon the other hand, the Irish, with, as a rule, excellent physique, generally die of phthisis within a short time of contracting it. Why? Because they have only recently begun to undergo tuberculosis selection, the huts or hovels in which they, for many generations, have lived in Ireland having never been air-proof. Further, until comparatively recent times, they were a rural, not an urban, people.

In course of time, as a direct result of the survival in the new and improved environment of Jewish infants inheriting the tubercular tendency, the mortality in the hygienic surroundings of the New World will probably equal the mortality in the unhygienic environment of the Old World. Congenital tendency toward tuberculosis—absence of resisting power to the attack of tubercle bacilli on the part of some portion of the body—appears to be a germinal variation to which the human family is markedly liable.

The medical profession for many years refused to admit that the tubercle bacillus, while the enemy of individuals, was the friend of the race, but at last the truth is becoming recognized. At the 1911 meeting of the Canadian Medical Association a prominent English (Liverpool) physician, Sir James Barr, made this assertion: "If you abolish the tubercle bacillus in Ireland, then it will certainly become a question of 'God save Ireland.'"

The *Medical Record* of March 18, 1911 (p. 493), in commenting upon the writer's tuberculosis statistics, said: "The decrease of

mortality from tuberculosis is probably not commensurate with the strenuous efforts which have been put forth to achieve this object." This statement is unquestionably accurate.

Moreover, to any one familiar with biology, our present methods of dealing with tuberculosis seem utterly inadequate,—first, because, in the majority of cases when the congenital predisposition is present, infection by tubercle bacilli is very difficult to avoid; [1] secondly, because persons of the phthisical type generally marry early in life, and beget children more rapidly than other people.—(Francis Galton.) Tuberculosis will automatically decrease when men and women possessing the tubercular predisposition refrain from giving birth to children. Compared with this remedy for tuberculosis, all other measures are likely to prove failures in the future, as they have done in the past.

[1] Prof. Karl Pearson's statistical investigation of 384 family stocks in which tuberculosis had occurred. (Publication II of Eugenics Laboratory, Dulau, London.)

THE FEATURES OF THE NORMAL HEART

BY PAUL BERGOUIGNAN, M.D.
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As a rule, medical students study the diseased organs, in the hospitals, without paying much attention to the normal ones and their various features in the healthy subject.

As regards the heart, there are few organs that present so many different aspects, owing to its anatomical form, that of the viscera around it, as well as to the shape of the thorax itself and the constitution of the person examined.

There is no question here of the slightest real alteration, which always shows that there is some organic or functional heart trouble, for we should always think that the slightest valvular murmurs (even where no lesion) are pathological, as well as the morbid variations of the intensity of the sounds, or of intervals between them, of heart rhythm, of heart speed (slow pulse or tachycardia), or even slight degrees of hypertrophy and dilatation.

We simply wish to call attention to some various results of the physical examination of the normal heart. They are of great interest to us, because it is easy to attribute a pathological reason to them when in reality they are normal.

First of all, what do we find in the examination of a *typical normal heart?* We see and can touch the impulsion of the heart's apex in the fifth intercostal space, half an inch inside the nipple line, or three inches from the mediosternal line. The surface of the impulsion can be covered by the end of the finger.

A strong percussion will limit the zone of absolute dulness between the apex and the left border of the sternum. A gentle percussion will give the zone of relative dulness, a rectangle-triangle, of which the base is the right border of the sternum and the summit the apex. The latter zone, whose area can be calculated, is the forward projection of the heart as it is seen in skiagrams.

To the ear, each cardiac revolution gives two sounds; the first dull and long, the second short and sharp. The silence following

the second sound lasts almost twice as much as that following the first sound.

Now, what are the chief *modifications that are not pathological of these normal sounds?*

First, very often, neither by sight nor by touch can we find the heart's apex. This *default of the heart's impulsion* is seen in emphysema cases, and also in those patients who have a wide and short thorax, with thick ribs and small intercostal spaces.

Next, we find an *exaggerated impulsion* over quite a large surface, both by sight and palpation. Sometimes at this spot we get a systolic retraction that is quite independent of any pericardiac lesion. These phenomena are seen in those subjects with a long thorax and wide intercostal spaces.

Cylindrical Thorax; False Hypertrophy of the Heart.—A patient comes complaining of heart palpitations and precordial sensations. He thinks he has heart-disease, and has perhaps been told by some doctor that he has a "little hypertrophy."

You find nothing special by auscultation, but, by inspection and palpation, the apex seems to beat very strong and very low down, inside the nipple and too near the left sternal border. However, the area of relative dulness is not increased, and you find the beat is in the fifth intercostal space, the normal one, while the ribs are separated by wide intervals. The thorax, in place of widening like a funnel upside down, on the contrary, lengthens and narrows from above to below. The costo-xyphoidian angle is quite acute, and the false ribs come down as low, in some cases, as the iliac crest.

These are the "cylinder thorax" forms that my late professor, Dr. Huchard, wrote about. Their history is simple. During adolescence, some crisis of rapid growth is localized in the bodies of the vertebræ; the thorax grows rapidly in height, while its contents remain stationary, and the plane of the ribs inclines vertically. When, later on in life, the heart and lungs develop, they try to adapt themselves to their cage. Hence palpitations, false hypertrophy, and, most often, wrong medical interference, prescribing rest and preventing proper exercise. If the patients, on the contrary, are taught proper respiratory movements and exercises, the chest will become normal. This therapeutic result is quite striking in young men, and we have seen it succeed in men of fifty years of age; but in many cases, as the years go on, the ossification of the cartilages tends to fix this

disease of growth, and the heart can really become hypertrophied. "Enlarge the thorax," said Dr. Huchard, "and the heart will not become enlarged."

It will only be necessary to mention the *false dilatations of the heart,* which will be observed sometimes when there is an atony and gaseous distention of the stomach; here the stomach balloons out and pushes the diaphragm up, sometimes as high as the fourth rib; it makes the heart horizontal, with its apex raised and carried out.

The heart-sounds often present, when normal, some *individual variations of timbre and intensity,* that depend upon the structure of the precordial covering, the state of the nervous system, and some other less definite causes. For instance: without hypertension or any cardiac or aortic alteration, the heart-sounds are often rough and more sonorous in nervous cases, making a "cardiac erethism."

The *cardiopulmonary souffles or extracardiac sounds* were well described by Professor Potain, and it is now known that they exist in the normal physiological state and are innocent curiosities of auscultation; but they are sometimes overlooked, and it is important to locate them and to explain to the patients who may be seared by them that they are harmless.

The *doubling up of the two heart-sounds* deserves a special mention, as this sign can be found in the normal state. As a rule, it disappears if the respiration is suspended for a moment.

The doubling up of the *second* sound seems to be due to a defect of synchronism at the aortic and pulmonary sigmoids. When heard at the base, it indicates a mitral stricture, according to Huchard. The sound that we mean is heard at the apex, where it might make one think of the mitral-opening-snapping described by Samson and Potain, in the stenosis of the orifice. In reality, it is a doubling up of the sound that is not pathological. It is clearer at the end of inspiration, and is frequently found in nervous people.

The physiological doubling of the *first* sound is heard still more frequently at the end of expiration. François Franck clearly explained the difference between this and the "bruit de galop." The systolic shock or first sound at the apex is followed by a dry, sharp click, and resembles the "bruit de trot" of Professor Lépine and the systolic gallop of Potain. Notwithstanding the teachings of physiology, it has been claimed to be caused by a defect of synchronism in the contractions of the two ventricles. Again, other writers

think that it shows a real cardiac trouble caused by a commencement of hypertension or arterial sclerosis.

In over 2,000 different observations we found the sign in more than 200 cases in nervous, neurasthenic, and dyspeptic people, as well as in healthy individuals. It coincides sometimes with the doubling of the second sound at the apex, and they both disappear or diminish during any arrest of respiration or after any effort, such as coming upstairs or walking fast. Any increase of nervousness or insomnia seems to increase it.

One might easily mistake this physiological doubling-up sound for the "bruit de galop," but we must remember that the former is always post-systolic and does not resemble renal gallop, which is always heard during diastole. It exists without tachycardia, and the quickening of the heart movements does not make it clearer, like the gallop, but rather tends to make it disappear altogether. If this doubling exists in albuminuria cases or in some doubtful hypertension, we should remember that it may be physiological, or at least nervous in character, and we shall not think at once of a renal gallop.

Most of these normal cardiac features are not easy of diagnosis and are often not thought of, when making a rapid examination of the heart. When, on the contrary, we are called to listen to the heart-sounds for a life-insurance case, or when anæsthetics are to be given, these normal variations may be mistaken for serious lesions, and this can lead to much trouble.

In such cases we must be careful not to inform the patient that he has a difference of any sort in his heart-sounds, even if we say it is of no importance. We should simply say that his heart is normal, or otherwise a nervous person, feeling his heart beat under a precordial wall that is too sensitive, will be sure to think something is wrong, and fear a serious heart lesion. This will end by consulting many other physicians and by none of them being able to convince him that he has no heart-disease. We may thus create what Huchard used to call "a disease that is all the more incurable because it does not exist."

Like him, we think that the initial error comes from the fact that we often attach too much importance to auscultation and percussion of the heart, without dwelling enough on the general and functional symptoms, which, after all, are our surest guides in diagnosis.

Surgery

ENUCLEATION OF THE TONSILS AND REMOVAL OF ADENOIDS AND OF THE LINGUAL TONSIL BY SIMPLE METHODS

BY CURTIS C. EVES, M.D.

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It is now almost universally recognized that the entire removal of the tonsil tissue is the best procedure where operative interference is advised, and the attention of the laryngologist is intent upon finding a safe and simple method which can be performed quickly, leaving a clean and uninjured throat.

It is undoubtedly better for each operator to select the best method at his command and then to perfect the technique to the best of his ability. After one method has been mastered, it is comparatively easy to make changes which seem best to suit the operator.

Nothing new or original is claimed in the following method. It is a simple combination of steps used by many different operators, and history tells us that the American Indian chiefs used to pluck troublesome tonsils from the throats of their tribes by means of the fingers long before they were molested by a civilized medical profession.

Long anæsthetization for operations upon the tonsils always more or less jeopardizes the life of the patient who is affected with hypertrophy of the lymphoid glands, and oftentimes it is necessary to remove the tonsils of patients suffering from heart, lung, or kidney complications to whom an anæsthetic is harmful. The average time required for this operation is about five minutes for the removal of both tonsils and adenoids. Where time is of paramount importance to the patient's welfare, it can be done in less than two minutes under nitrous oxide, ethyl chloride, or primary ether.

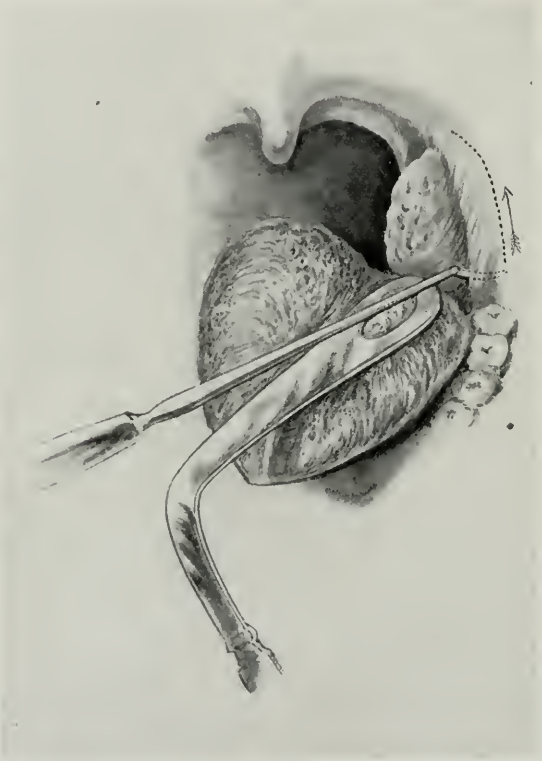
I have used the following technique in a series of over seven hundred cases of all kinds of tonsils without any serious accidents.

General anæsthesia (ether) is preferred. After the patient is

thoroughly under the influence, the anæsthetic is stopped. Further anæsthetization is rarely ever necessary.

The patient is then placed on the operating table in the horizontal position, with a small sand pillow underneath the shoulders, causing the head to incline slightly backward. The mouth-gag is introduced in the ordinary manner, the tongue depressor is placed well back

Fig. 1.



The right-angular knife introduced underneath the anterior pillar preparatory to making the incision upward. The incision is terminated by rotating the knife inward, bringing the end out in the supratonsillar fossa.

upon the side of the tongue, and traction is made upon it downward and to the opposite side of the mouth, thus putting the outer pillar on the stretch and bringing it prominently into view. A right-angle curved knife is now introduced below and carried upward underneath the anterior pillar, between it and the capsule of the tonsil, separating the fold of mucous membrane reflected from the pillar upon the capsule (plica tonsillaris) and any adhesions that have

formed between the anterior pillar and the tonsil; the incision is terminated in the supratonsillar fossa (Fig. 1). A knife with the opposite curve is now introduced with back uppermost into the top of the incision and gently held above and pushed backward over the superior pole of the tonsil; it is then rotated inward, cutting between the upper part of the posterior pillar and the capsule (Fig. 2).

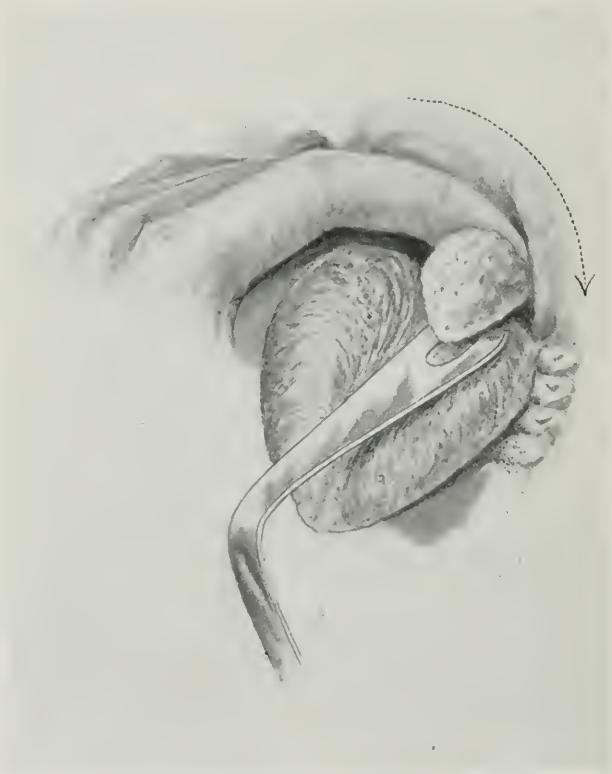
Fig. 2.

The left-angular knife separating the upper end of the posterior pillar from the capsule of the tonsil. The knife is introduced with the back upward, then rotated inward, making an incision about the length of the knife-blade.

The ball of the index finger of the left hand is placed in contact with the capsule upon the superior pole of the tonsil underneath the junction of the two incisions, and by gentle pressure downward, together with a rotary motion outward and downward, the finger forces the tonsil in its capsule out of its fossa. This motion of the finger is kept up until the fibrous tissue holding the capsule is entirely

separated except at the base, where it is usually much thicker and more firmly attached (Fig. 3). The tonsil now lying loosely in the throat, except at the base, is caught over the upper part by a tonsil forceps, and is held on the stretch by an assistant, while the operator slides the loop of the snare over the forceps and fits it around the fibrous base with the left forefinger, at the same time gradually tight-

Fig. 3.

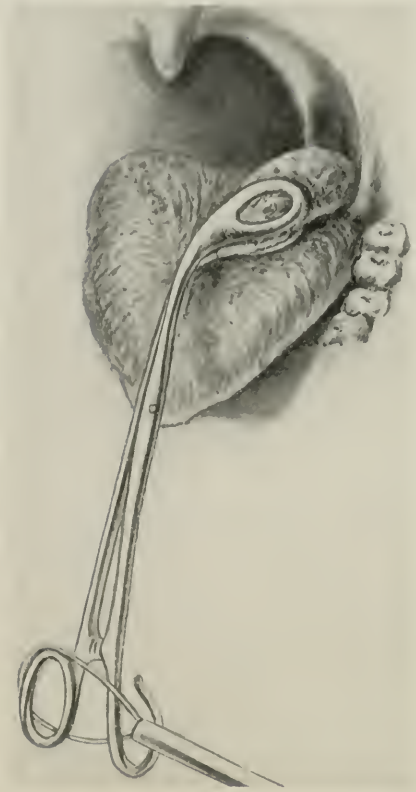


Finger peeling the tonsil in its capsule out of the fossa.

ening the loop with the right hand (Figs. 4 and 5). When the loop is adjusted, the left hand assists the right in making the cut through the base and the tonsil is removed.

The pillars of both tonsils are separated with the knives before the finger is used, then the removal of one tonsil is entirely completed. If both tonsils are peeled out with the finger before either base is snared off they are very apt, especially if large, to fall inward,

Fig. 4.

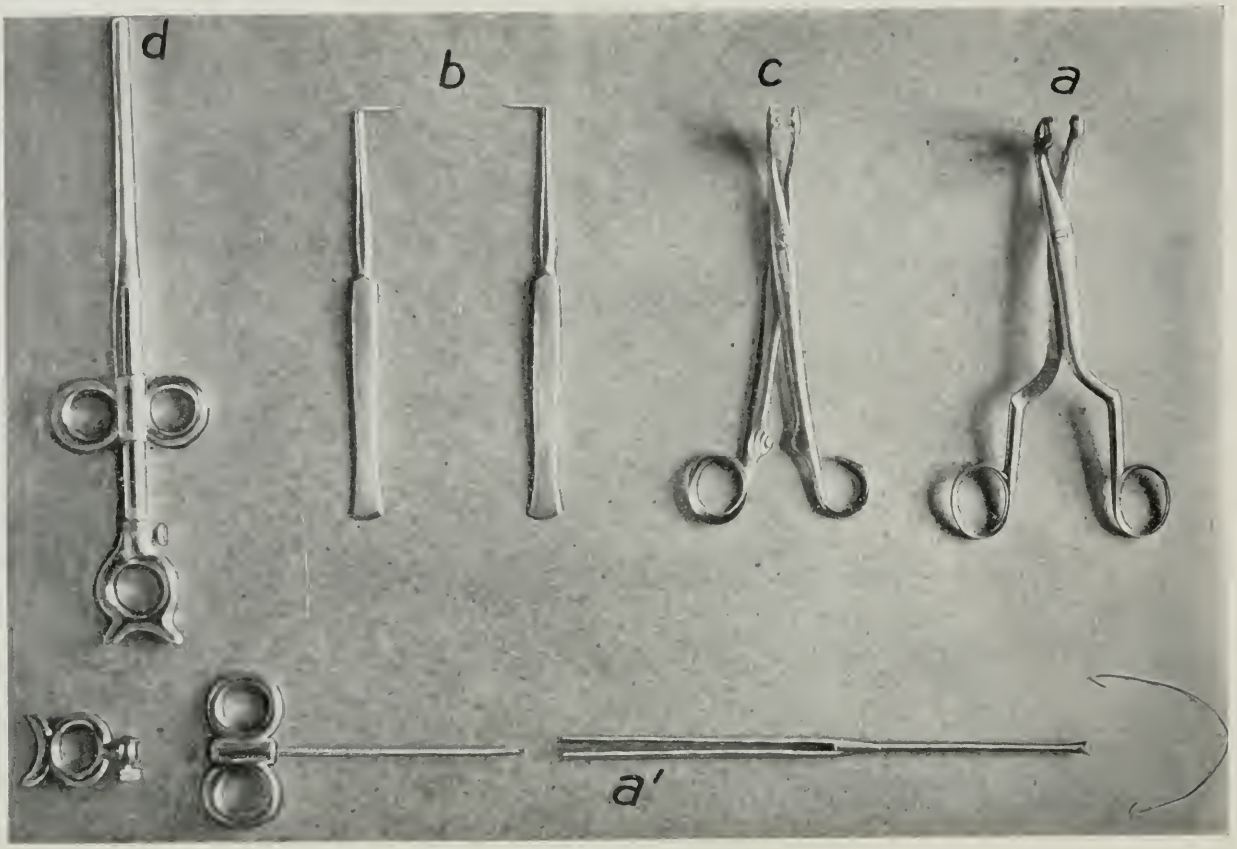


The forceps grasping the tonsil, and loop of the snare being passed over the handle.

Fig. 8.

Method of removing the lingual tonsil by the Miles lingual tonsillotome.

Fig. 6.



Special instruments used:

a. Eves tonsil snare.
a[1]. Eves tonsil snare in parts.
b. Eves modification of Leland's tonsil knives. The blades are ground shorter and rounded upon the points.
c. Eves tonsil forceps.
d. French adenoid forceps.

forming a valve-like closure of the larynx which may severely interfere with the respiration of the patient.

The field is kept clear of blood by the assistant with gauze sponges. As soon as both tonsils are removed the patient is turned upon the side with the head low to allow the blood to drain from the mouth. Very little bleeding usually occurs. If bleeding continues it is most often

FIG. 5.

Snare fitted around the base of the tonsil.

due to faulty technique, where the pillars have been cut, or when a piece of tonsil at the base has been cut through by not properly fitting the snare around the tonsil.

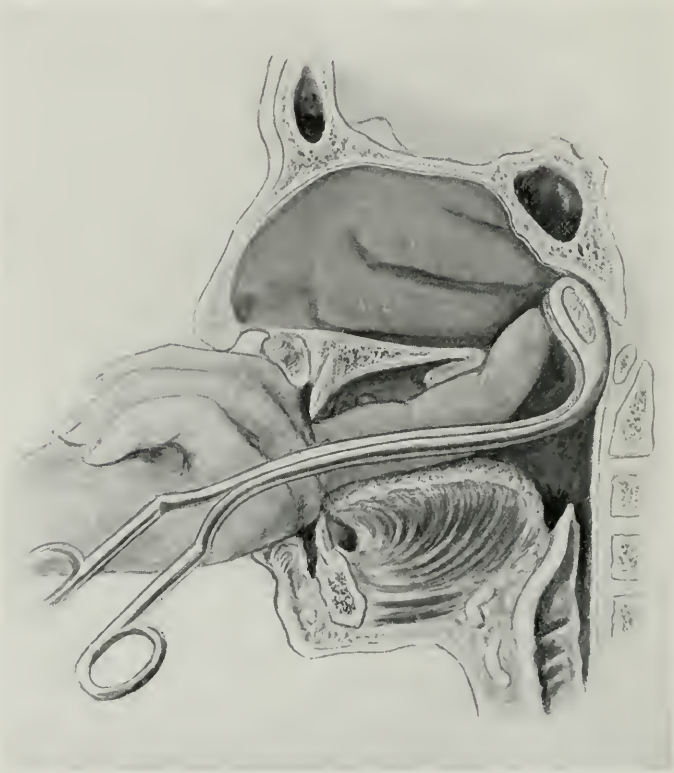
The instruments used are seen in Fig. 6.

The tonsil may be entirely taken out by the finger without the use of the snare, but usually so much force is necessary to tear through the fibrous tissue at the base that the reaction is much greater than by snaring through it.

The adenoids are removed, while the patient is turned upon the side, in the following manner:

The left forefinger holds the uvula and soft palate forward, the tip of the finger is placed in contact with the septum and is used as a constant guide for the forceps (Fig. 7). The forceps are placed

Fig. 7.



Removal of adenoids with the French forceps, showing the position of the finger and the forceps grasping the adenoid mass.

into the nasopharynx with the blades closed, in the median line, so that the end comes flush with the posterior margin of the septum. The blades are then opened, pushed firmly backward over the growth, closed tightly and with a twisting motion made to partly cut and partly tear the adenoid from its attachments. If any pieces remain they are grasped with the forceps and twisted off. This is followed by curetting the surface with the end of the finger wrapped with

several layers of thick-meshed gauze saturated with pure alcohol. By this method the adenoid vegetation is thoroughly removed, injuries to the posterior tip of the eustachian tube and stripping downward of the mucous membrane of the posterior pharyngeal wall are avoided. The alcohol disinfects the wound and considerably reduces the bleeding.

To complete the tonsillar groups in the throat we have yet to mention the lingual tonsil. It being the least considered and most often overlooked, I shall treat its hypertrophy a little more fully than the other two groups, of which only one method of surgical treatment has been considered.

The lingual tonsil is an ill-defined mass of lymphoid tissue found at the base of the tongue, behind the circumvallate papillæ, in front of the epiglottis. Normally, we see several small, irregular islands of lymphoid tissue about the size of a split pea on either side of the median line. When hypertrophied the appearance presented is not unlike that of the pharyngeal tonsil, except that it is of a brighter red color and more nodular. Only two or three of the islands may become hypertrophied, projecting backward in wart-like growths. Frequently the growth is covered with varicose veins, or the varicosities may occur unaccompanied by hypertrophy, when the condition is known as lingual varix. The causes of hypertrophy of the lingual tonsil are not well known. It is, however, a well-known fact that hypertrophy usually begins about the time of life that the pharyngeal tonsils and adenoids begin to undergo atrophy, and that it does occur much more frequently in nervous, highly-strung females is an undisputed fact. Dudley, in his article published in the *Transactions of the American Laryngological, Rhinological, and Otological Society*, states that nine-tenths of the cases are seen in females after the twelfth year. He also says that trauma, rheumatism, gout, constipation, and indigestion are predisposing causes. Lingual varix is most frequently seen in elderly people, and it then rarely gives symptoms. A hypertrophied lingual tonsil pushes forward against the epiglottis, and may press so against it as to entirely envelop the tip, obscuring it from view.

The most annoying symptoms are caused by mechanical contact with the epiglottis. They are,—a sense of fulness or lump in the throat (often erroneously interpreted as globus hystericus), a tickling sensation, and a dry, irritating cough, worse when lying down.

Patients are apt to complain of some interference with swallowing. In singers, hoarseness and loss of voice are common, due to overdevelopment or fatigue of the muscles of the larynx, caused by clearing the throat and coughing. Among the less frequent symptoms are reflex pains and asthmatic attacks. When laryngeal varix accompanies hypertrophy, the cough is apt to be followed by bloody expectoration, due to rupture of some of the veins. These patients are usually much alarmed, fearing they have pulmonary tuberculosis.

The diagnosis is made and treatment carried out by use of the laryngeal mirror. The patient holds the tongue forward with a napkin between the forefinger and thumb of the right hand. The mirror is placed gently against the soft palate so as to bring the reflected image of the growth clearly into view (Fig. 8). The following treatment has given me the best results:

Where the symptoms are of short duration, applications of 2 per cent. cocaine solution, followed by 30 per cent. argyrol in equal parts of glycerine and water, are made every other day. Should the growth not become reduced in size and the symptoms subside after a few treatments, where the growth is of a soft nature, it is cauterized with 50 per cent. silver nitrate. Two parallel lines are made, one on either side, through the most prominent parts of the growth, 10 per cent. cocaine being used beforehand. Care should be taken not to touch the epiglottis or allow any of the silver solution to trickle into the larynx.

When the growth is large and of a denser nature, surgical removal is imperative and is carried out as shown in Fig. 8. A 10 per cent. solution of cocaine is applied two or three times, the Miles lingual tonsillotome is carefully fitted over the most prominent part on one side and made to cut through by pulling the knife-blade outward, at the same time pushing the forked blade inward. The other lobe is removed in the same manner. Sometimes it is necessary to make more than one cut on either side. The tissue is more fibrous than pharyngeal tissue and requires some force to make the instrument cut through. The instrument should be held firmly against the base of the tongue until the full cut is made. Very little bleeding occurs. The surfaces are then painted with 3 per cent. silver nitrate. Where lingual varix occurs only the cautery should be used, and repeated ever four or five days for several times.

THE PROGNOSTIC VALUE OF AN IMMEDIATE EXAMINATION OF PERITONITIC EXUDATES*

BY D. P. D. WILKIE, M.CH., F.R.C.S.
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When the surgeon has concluded an operation on a case of diffuse peritonitis, and is faced by his next duty, that of giving a prognosis, he is frequently forced to be somewhat guarded in his opinion. Although, from a more or less extensive experience of such cases, he may gauge fairly accurately the patient's chances of recovery, he reflects that the infection may be more virulent than he judges it to be, and in giving his opinion he falls back on the dictum "that the first forty-eight hours will tell, but until that time is over there must be some anxiety as to the patient's condition." Any reasonably simple yet scientific method whereby he could gauge the virulence of the infection and the vigor of the patient's resistance, and could thus be in a position to give a more confident prognosis, would be of undoubted value and would save much needless anxiety.

It was hoped that by investigating the bacteriology of peritoneal infections some data of prognostic value might be obtained. A vast amount has been written on the subject of the bacteriology of peritonitis, and the statistics given and the views expressed by various writers have been so conflicting that the practical surgeon has come to regard the bacteriological investigation of cases of peritonitis as being of little real value in influencing the prognosis in any given case.

The skeptical attitude which so many surgeons have taken up towards the practical worth of the bacteriologist's findings cannot be wondered at when the clinical course of their cases is so frequently at variance with the expectations of the bacteriologist. Although infections of the peritoneum by the streptococcus are gen-

* The experimental work referred to in this paper was carried out in the Laboratory of the Royal College of Physicians, Edinburgh. The clinical observations were for the most part made in the clinic of Professor Caird, in the Edinburgh Royal Infirmary.

erally more serious than those by the *Bacillus coli communis,* it is by no means rare to see recovery in a case of streptococcal infection, or, on the other hand, a rapidly fatal issue in a case of pure infection by the colon bacillus. Moreover, the time required for a bacteriological examination in itself robs this method of what practical value it might otherwise have. The truth of the matter lies in the fact that pure bacteriology deals with only one side of the question, and the important point for the surgeon and patient is not the name or the cultural characters of the invading germ, but its relative virulence for the particular individual. It is on this personal equation that the surgeon wishes light in deciding his treatment and in giving his prognosis.

By careful microscopic examination of stained films of the peritoneal exudate one can gauge fairly accurately what is the relation between the two important factors, viz., the patient's resistance and the virulence of the bacteria for the patient. Such an examination can be made in the operating theatre in a few minutes; the film can usually be stained and examined long before the surgeon has completed his work, so that he may also have the benefit of such accurate information as it affords in deciding the question of drainage.

After three and a half years' experience of this method of immediate microscopic examination of the exudate in cases of free peritonitis I am fully convinced of its value.

From a prolonged study of films from experimental peritonitis in animals I found that it was possible from a glance at a film made from the peritoneal fluid not only to foretell whether the animal would recover or would die, but, in the latter case, to gauge fairly accurately the number of hours it would live.

We know that as a rule when bacteria invade the peritoneal cavity fluid is poured out, and for the first two or three hours there are but few cells in the exudate. During this early stage the bacteria disappear to a great extent from the exudate; many of them undergo lysis, while great numbers adhere to the omentum. At the end of three or four hours the character of the exudate changes in two respects, viz., the bacteria are again much more numerous and the polymorphonuclear leucocytes are present in great numbers. Thereafter, unless the protective mechanism of the animal is paralyzed

by the virulence of the bacteria, the polymorphonuclear leucocytes continue to arrive in increasing numbers until the bacteria are overcome.

When the animal's resistance is relatively high these polymorphonuclear leucocytes, or microphages—to use Metchnikoff's name for them—rapidly ingest the bacteria. If film preparations be made from the peritoneal exudate during this stage of active and successful phagocytosis the polymorphonuclears are seen to stain well, many of them contain ingested bacteria, and their nuclei are clear and well defined. In such a favorable case, few, if any, extracellular bacteria are to be seen. In colloquial language, the cells are obviously winning.

When the animal's resistance is low or the bacteria are specially virulent, the polymorphonuclears do not appear in such large numbers, and, though some of them may contain bacteria, many show signs of degeneration, and extracellular bacteria are seen in the film.

In cases where peritonitis has existed for twelve hours or more, and where the animal is going to recover, the peritoneal films are characteristic: numerous healthy polymorphonuclears are seen, some of them containing bacteria, but the striking feature of the film is the presence of the large mononucleated macrophages. Some of these cells may have ingested bacteria, some of them have engulfed one or more of the microphages.

The origin of these large mononucleated phagocytes is still uncertain; some of them may arise from the mononucleated cells of the blood, many of them probably come from the endothelium of the capillaries, while others arise from the endothelium lining the peritoneal cavity. Although the omentum may constitute a fruitful source for these macrophages, it is probably less important in this respect than many writers have indicated. I have repeatedly found that removal of the omentum, although it undoubtedly lowers an animal's resistance to peritonitis, does not in a case which is recovering, appreciably affect either the time of advent or the number of the macrophages in the peritoneal fluid.

The appearing of the macrophages and the display of their phagocytic activity for microphages is invariably a favorable sign, and when in a given case the large mononucleated cells outnumber the polymorphonuclears and have ingested many of these cells, we

realize that the process of repair is in full swing, and that recovery is assured. In such a case the exudate is usually thick and purulent, yet drainage of the peritoneal cavity is not only unnecessary but is meddlesome.

Peritonitis, as we meet with it in the human subject, differs from experimental peritonitis in that it usually spreads from a local focus and is not immediately generalized. There are, however, a very large number of cases where the surgeon is called on to treat a free and diffused, though not generalized, peritonitis and in this class of case an immediate and accurate scientific index of the relative strengths of the infection and the reaction is of great value. Certain common-sense precautions must be taken in using this form of examination. In the first place, it is obviously of no value in localized collections of pus which are walled off by peritoneal adhesions; here the pus may be swarming with bacteria and recovery ensue. Nor is it of much value when applied to the reaction fluid in the peritoneal cavity surrounding a large walled-off abscess, for here the fluid may show healthy cells and few or no organisms and the patient may yet die from toxæmia from the large localized purulent collection. If, however, the test be applied in cases of free diffuse peritonitis, and the drop of fluid for examination be taken from the general exudate, a very important and a very reliable index of what is happening may be obtained.

TECHNIQUE OF THE EXAMINATION.

This is eminently simple, and can be carried out by any theatre assistant or nurse.

When a case of suspected peritonitis is to be operated on there should be in the theatre a microscope with oil-immersion lens, some glass slides, a platinum needle, a spirit lamp, and some simple stain, such as thionine blue or polychrome-methylene blue.

As soon as the peritoneal cavity is opened a drop of the escaping exudate is taken on the sterilized platinum needle, smeared on a slide, and dried over the spirit lamp. The film is then stained for two minutes, washed, rapidly dried, and examined under the oil-immersion lens. While the preparation of the film must be deputed to an assistant or nurse, the examination and interpretation of it should, where possible, be done by the surgeon himself. The inter-

Fig. 1

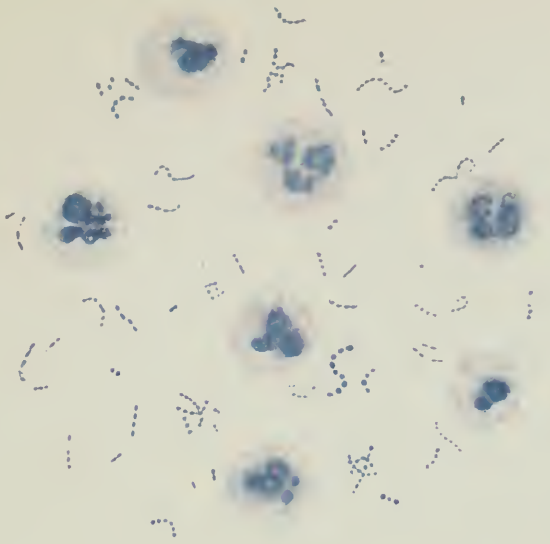

Fig. 2.

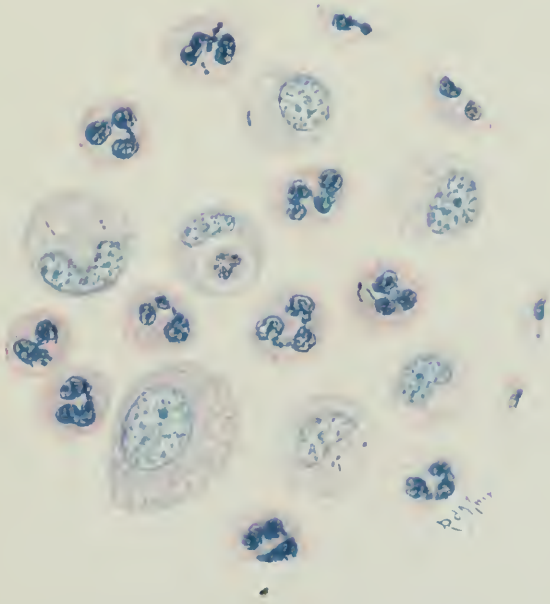

Fig. 1.—Fatal streptococcal peritonitis: no phagocytosis; faint staining.

Fig. 2.—Film from a case of general peritonitis: successful phagocytosis; numerous mononucleated macrophages

ruption of the operation for a few moments to glance down the microscope is more than compensated for by the accurate knowledge which the operator obtains of the nature of the fluid with which he is dealing.

This method is so simply carried out that it should not be confined to the larger hospitals and university clinics, for it can be as readily employed in the cottage hospital and even in the private house, and I consider that a portable microscope should be one of the regular articles of the surgeon's emergency bag.

The accompanying figures are from drawings of slides made at operations on six cases of peritonitis.

Fig. 1 is a drawing from a film made from the exudate in a case of delayed postoperative peritonitis. The case was that of a woman who three weeks before had been operated on for a pancreatic cyst. The cyst had been treated by marsupialization and the patient progressed well for twenty days. She then developed the signs of peritonitis, and accordingly the abdomen was reopened. On incising the peritoneum a considerable quantity of turbid serous fluid escaped, and a drop of this was immediately examined microscopically. Despite the fact that the general appearance of the patient did not suggest that the case was hopeless, a fatal prognosis was given as soon as the film was examined. The absence of phagocytosis, the commencing degeneration of the leucocytes as shown by faint staining and nuclear fragmentation, along with the swarms of bacteria free in the fluid, led one to foretell that death would occur within sixteen hours, and in spite of all that could be done this was the case.

Fig. 2 is from a film made from the peritoneal exudate in the case of a man, æt. 25, who was admitted to hospital suffering from generalized peritonitis secondary to acute appendicitis, the acute symptoms having started four days previously. On admission he appeared markedly toxæmic and very ill. His abdomen was greatly distended and was tender and rigid all over. Immediate operation was undertaken, the abdomen being opened by a free incision in the mid-line. Seropurulent fluid escaped, and a drop of this was at once taken for examination. The coils of small intestine were greatly distended and covered with flakes of lymph. The appendix, which was gangrenous, was removed and a quantity of sterile vaseline oil was poured into the abdomen to delay the formation of plastic adhesions,

and thus to favor peristalsis and drainage. A pelvic drain was inserted and the median wound closed. The film revealed the presence of active phagocytosis, of numerous mononucleated macrophages, of commencing phagocytosis of the polymorphonuclears by the large mononuclears, and of practically no extracellular bacteria. Therefore, in spite of the unfavorable appearance of the patient and the generalized nature of the peritonitis, a hopeful prognosis was given. This was justified, as the patient progressed favorably and left the hospital well fifteen days after operation.

Fig. 3 is from the case of a woman, æt. 25, who developed the signs of acute appendicitis five days before her admission to hospital. She had been kept in bed with fomentations on the abdomen and given a low diet, and for four days, although the pain had persisted, her symptoms had given rise to no alarm. On the fifth day, however, twelve hours before admission to hospital, she had been suddenly seized with very acute pain generalized over the whole abdomen, had vomited several times, and her pulse, which previously had been little above the normal, had rapidly risen in frequency. On admission to hospital she looked ill and her pulse-rate was 140. The abdomen was rigid and tender all over, and it was obvious that she was suffering from general peritonitis. Immediate operation was undertaken, and on opening the abdomen by a mid-line incision seropurulent fluid with a slightly fœtid odor welled out. A drop of this was taken for immediate examination.

The appendix was found greatly inflamed, with a gangrenous portion near its tip and a large perforation alongside this. It was removed and the stump invaginated. Before this had been completed, however, examination of the film from the exudate showed that the case was hopeless. The absence of phagocytosis, the imperfect nuclear staining of the polymorphonuclear leucocytes, and the large number of free extracellular bacteria clearly showed that the germs had gained the upper hand and that the protective mechanism was becoming paralyzed. In spite of free drainage and intravenous saline infusion, the patient died seventeen hours after operation.

Fig. 4 is from the case of a boy, æt. 11, who was admitted to hospital for operation forty-eight hours after the onset of an acute attack of appendicitis. For the first twenty-four hours of his illness he had suffered from moderately severe pain in the right side of his

Fig. 3

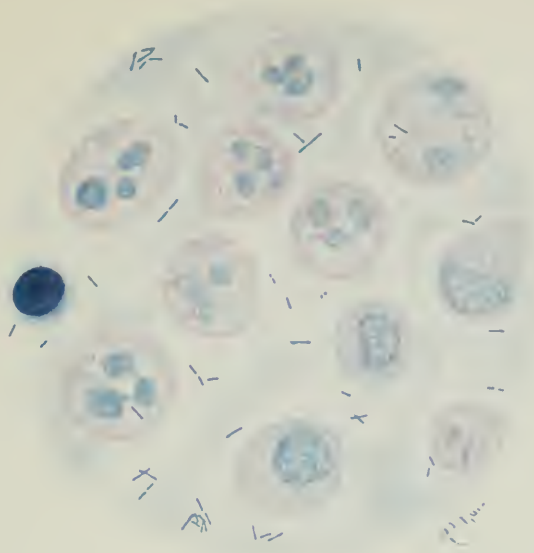

Fig. 4.

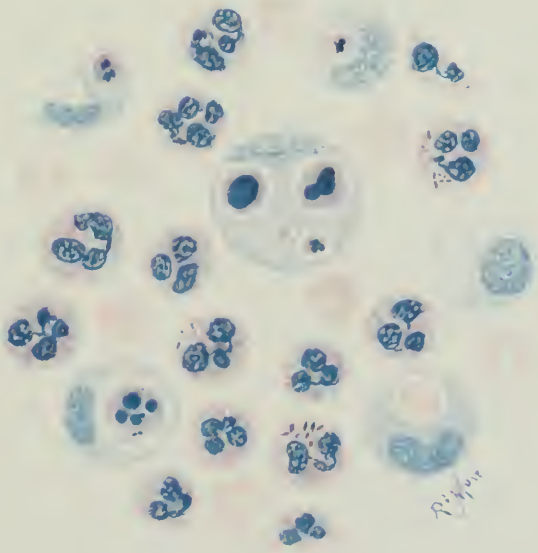

Fig. 3.—Fatal peritonitis; absence of phagocytosis; imperfect nuclear staining of leucocytes.

Fig. 4.—Diffuse peritonitis from perforative appendicitis; no bacteria free in fluid; phagocytosis of polymorphs by macrophages; recovery assured.

abdomen. At the end of that time, however, he had been given a dose of castor oil, and shortly thereafter he was seized with very severe pain all over the lower part of the abdomen. On admission to hospital he presented the signs of a diffuse peritonitis, the abdomen being fixed in its lower half and very tender. The abdomen was opened without delay by the gridiron incision in the right iliac region. Seropurulent fluid escaped in quantity, and a partially gangrenous and perforated appendix presented. A suprapubic stab-wound was made and a glass drain inserted, quantities of purulent exudate at once escaping from the pelvis. A drop of this fluid was taken for immediate examination, and meanwhile the appendix was removed. Before the operation was completed a very favorable prognosis was given, for the film showed the presence of a good cellular reaction, active phagocytosis, and numerous macrophages already ingesting the polymorphonuclears—in short, that the infection was already well-nigh overcome.

The boy left hospital in thirteen days with a healed wound.

Fig. 5 is from the case of a man, æt. 54, who was admitted to hospital for operation five days after the onset of an acute attack of appendicitis. On admission he appeared to be in tolerably good condition; his pulse-rate was 104, temperature 99° F. The abdomen was rigid and tender below the umbilicus, especially on the right side. There was no tenderness in the upper part of the abdomen. Rectal examination detected fulness and elicited tenderness in the pouch of Douglas. He was at once taken to the operating theatre and the abdomen was opened by the gridiron incision. Purulent fluid was found free in the lower part of the abdomen. A suprapubic opening was made and a large quantity of seropurulent fluid evacuated from the pelvis. A drop of this was taken for examination. The appendix, which was gangrenous in its distal half, was then dealt with. The film revealed the presence of very numerous bacteria, which were for the most part extracellular; phagocytosis, although active, was obviously inadequate, and many of the leucocytes showed degenerative changes. A bad prognosis was consequently given. In spite of proctochysis, stimulation, and intravenous saline infusion, the man died two and a half days later from a spreading peritonitis.

Fig. 6 is from the case of a woman, æt. 25, who, for two days

before admission to hospital, had suffered from severe pain in the lower part of the abdomen. Seen shortly after admission, she looked ill, though her pulse was not much accelerated. There was marked tenderness and boarding of the lower half of the abdomen. Her history suggested tubal trouble, and accordingly the abdomen was opened by a median incision below the umbilicus. The coils of small intestine which occupied the pelvis were coated with lymph, and there was a quantity of purulent fluid in the pouch of Douglas. A drop of this was immediately examined. Similar purulent fluid was seen issuing from the ostium of a much-inflamed left Fallopian tube, which was thereupon excised. The film showed a complete absence of bacteria, numerous large macrophages ingesting the polymorphonuclear cells—in other words, all the signs of a healing peritonitis, and that, therefore, the abdomen might be closed without drainage.

Fig. 5.

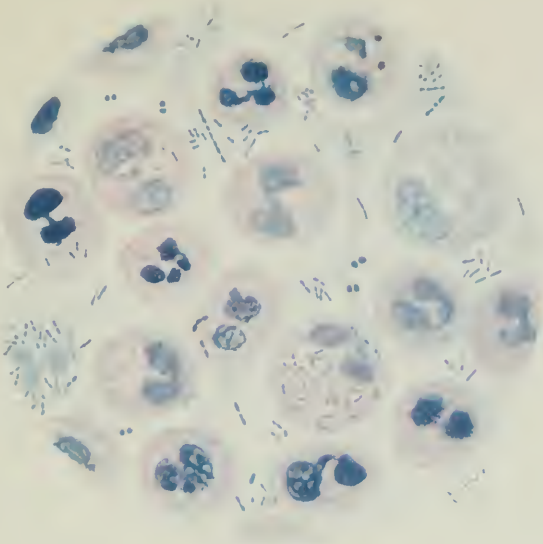

Fig. 6.

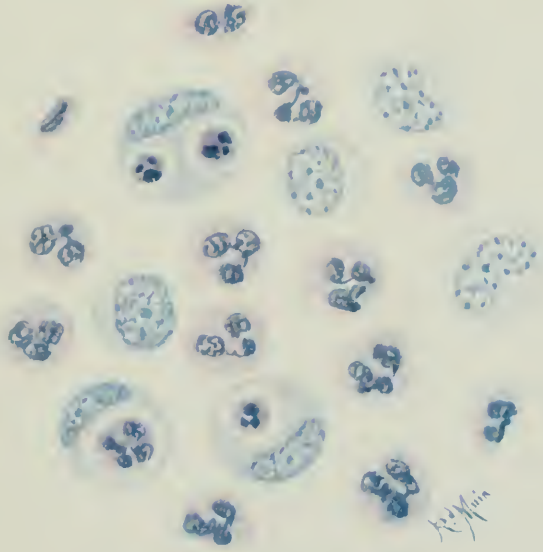

Fig. 5.—Diffuse peritonitis from appendicitis; active but inadequate phagocytosis; fatal.

Fig. 6.—Film from pus in pelvis; recovering peritonitis; no drainage required.

WEAK FEET*

BY IRVING DAVID STEINHARDT, M.D.*

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New York City

No, I am not apologizing for the subject I have chosen for my present paper, for every day convinces me more and more that for some unexplained reason practitioners "fall down" in properly diagnosing the weak foot. There are also many who make the diagnosis properly, but fail absolutely in giving the proper treatment. After making the diagnosis, your order to the patient to go to the most convenient dry-goods store, shoe store, or drug store and to purchase a pair of "alias" arch supporters is almost on a par with the doctors who turn their baby-feeding cases over to the manufacturers of infant foods to solve the feeding problems for them.

Your patient wanted not only a correct diagnosis, but correct treatment, and most feet need preparative treatment even before a properly made and fitted arch supporter can be worn. Therefore you can readily see why such an order is poor treatment. In answer to this many of you will say, "No patient of mine has ever come back to complain about the inefficiency of these arch supporters." You are quite correct, too, in making this statement. The only trouble is that it is only too true. The patients did not come back to you; they carried their troubles to someone else and stated that your treatment has "done" them—no good. Every one of us who do this work can show you at our offices and clinics every year thousands of patients who have been treated in this way without relief and come to us seeking our care and attention. To say the condition of weak foot is entirely overlooked thousands and thousands of times is, I believe, absolutely a truthful statement.

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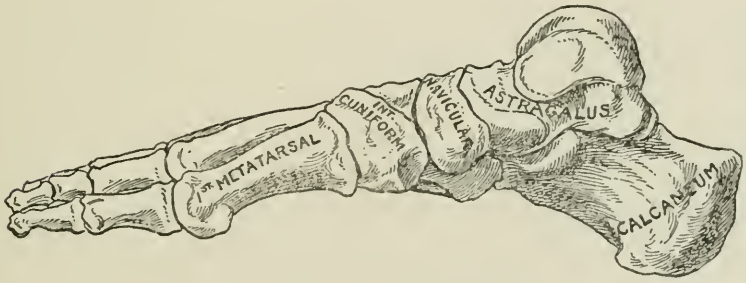
* A summary of papers read before the Norfolk County Medical Society, of Norfolk, Va., and the Medical Society of Greater New York, June, 1912.

Weak foot presents a marked variety of symptoms, many of which are far removed from the vicinity of the feet, so that may account for some of the incorrect diagnoses. Rheumatism—that much-abused and misused term—is the favorite misdiagnosis in cases with direct foot symptoms. I sometimes wonder what ever the profession would do without this convenient "hitching post" to tie various numerous symptoms in all parts of the body to at times. Of course, the numerous various rheumatic cures do not relieve cases of weak feet, so cases thus wrongly diagnosed are never relieved by these treatments, nor are your patients grateful to you for taking their money for treating them for something they never had, particularly if you have used the salicylates and upset their stomachs —something that the salicylates are very prone to do, as you know. Rheumatism and other arthritic conditions may complicate the condition of weak feet, and must not be overlooked when they exist in conjunction with it, but neither must the weak feet be overlooked as a condition of no importance because the other conditions exist. I dwell so much on these apparently trivial considerations because they are not so trivial as they seem. They are important to know and to remember at the proper time. It is really absolutely amazing to know how many times the orthopædic surgeon is called upon in consultation to diagnose some supposedly rare or difficult condition which has resisted all medication, and finds nothing more serious that one of the several varieties of weak feet. We may call it by a fancy name to save the physician, and speak of "pes planus" or "pes planus dolorens," but it is nothing more than weak feet, nevertheless.

To define weak foot is simple enough. Unfortunately, most of us, in speaking of it, have gotten into the habit of calling it flat-foot. This is misleading, and is perhaps the cause of the condition being overlooked so often. The examiner expects to find a foot or pair of feet with no arch at all. In a real case of flat-foot this is so, but bear in mind that while every case of flat-foot is a case of weak foot, not every pair of weak feet are flat feet. When the ligaments of the tarsal bones forming the arch of the foot weaken, the result is that they stretch, the degree of stretching depending upon the amount of strain causing the weakening and the strength of the ligaments, and this stretching is the primary cause of the weak foot.

This weakening of the ligaments causes an additional amount of work to be thrown on the supporting muscles. These are in turn unable to take up properly the extra work put upon them. Their failure to accommodate themselves to the changed conditions is the secondary cause of weak foot. The superincumbent weight of the body is carried upon two uprights or stilts, if we may so designate the lower limbs above the tarsal bones, the under surfaces of the tibia and fibula articulating with the astragalus, and the astragalus rests upon the os calcis or heel bone, while it is joined to the navicular bone in front. With the weakening and stretching of the binding ligaments followed by the giving away of the supporting muscles we have a partial or complete obliteration of the arches of

FIG. 1.



The bones of the foot—internal aspect.

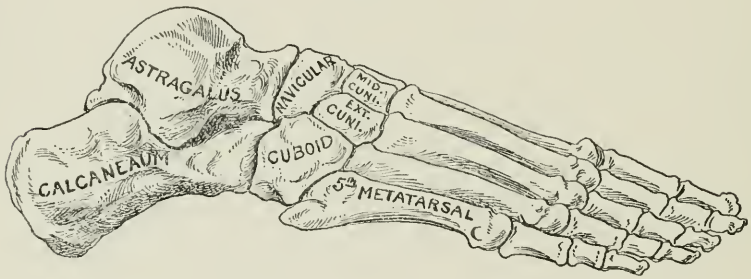
the feet, with a varying degree of eversion of the foot. This eversion may be most marked or hardly perceptible, and you will have cases showing every degree within these two extremes. Many postfracture weak feet show very marked degree of eversion.

The causes of weak feet are many—so many that one hesitates to attempt to chronicle them all. I shall, however, try and give you the main causes. Perhaps about the largest contributing cause is bad shoes. By bad shoes we mean any shaped shoes that do not conform to the lines of normal feet. Shoes, contrary to the dictates of those who make them, should be built to fit the feet, and not the foot forced to fit the shoes. So long as ill-fitting, abnormally-shaped shoes are purchased by the general public, just so long will weak feet increase. From sixty per cent. to seventy per cent. of the people are affected with this condition. Under bad shoes must be included those with those silly high heels that have become so

popular, likewise the so-called short vamp shoes which squeeze the feet in length and breadth both. Button shoes give less support than do lace shoes. Low-cut shoes are also classed among the offending ones. These shoes tend to throw the feet into improper positions that change the proper distribution of weight. The strain thereby put on the feet causes a weakening and stretching of the ligaments primarily and then the muscles of the bones of the arches. Result: weak feet.

To those who like to wear a sensible shaped low shoe I would say, develop and strengthen the ligaments and muscles of your feet to a proper degree, and then the lack of support in the low shoe will not be missed. Again, you all remember your school days when

Fig. 2.



The bones of the foot—external view.

you were told to stand with heels together and toes apart. Our drillmaster must have had a grudge against us, for this was certainly a fine way of standing to ruin our feet. Nowadays we know a correct way of standing is in a straight line, and perhaps the posture that would put the least strain on our arches would be one in which the toes were in what is commonly called a "pigeon-toed position."

Lack of development of the lower limbs is another cause. Many take special pains to develop their arms and chest and never give a thought to the abdominal muscles and those of the lower limbs. General debility, anæmia, wearing of "sister" shoes, obesity, slipper wearing, or walking much in the bare feet are some additional causes of weak feet. Ill-fitting stockings are most harmful. Occupations which require practically constant standing, stair climbing, the use of ladders, jumping, etc., are all very hard on the feet. Tile and stone floors are also factors. Postmen, waiters, bookkeepers, sales-

people, canvassers, clerks, bank tellers, conductors, etc., are very likely to be affected. However, the rich and poor in every walk of life, whether in work or play, furnish us our cases, and you must be ready to diagnose it in the prince or the pauper, the social butterfly or the worker. Both sexes seem to be equally affected and likewise all ages from early childhood to senility. In the negro race the condition is almost to be spoken of as normal, yet it is not usually a source of pain, trouble, or disability to them, because of the long time it has existed in that race.

Weak feet may be divided into several divisions for the sake of classification, although the symptoms of all are practically the same, merely accentuated by the severity of the case. My classification is as follows: (*a*) simple weak foot, a condition in which testing for the normal arch shows a varying degree of "dropping of the arch" with practically no eversion or stiffness; (*b*) simple flat-foot, which is merely the preceding variety, except that the "dropping of the arch" is complete; (*c*) everted weak foot: this is merely class *a* plus eversion of varying degree; (*d*) everted flat-foot, which is class *b* plus eversion; (*e*) rigid everted weak foot: this class is explained by its name, and the "dropping of the arch" may be complete or incomplete; (*f*) rigid flat-foot: in this class there is marked rigidity, with deformity and eversion; (*g*) traumatic weak foot, a self-explanatory name, in which the arch may or may not be entirely obliterated: this variety is usually found after fractures, sprains, and operations; (*h*) paralytic weak foot, another self-explanatory division, and usually found in conjunction with infantile paralysis and other paralytic affections in which the lower extremities are affected.

The complications of weak feet are important. One of the most important are varicosities of the veins, both deep and superficial, of the leg. In March, 1909, in a paper published in the *New York Medical Journal,* entitled "Painful Heels," I showed the relation between hypertrophy of the tubercle of the os calcis and weak feet. This is a most painful complication, which at times requires operation to completely relieve; chronic inflammation of the lower epiphysis of the tibia causing a thickening of this end of the bone. Gonorrhœal rheumatism in the subacute stage, arthritis of

various kinds, rheumatism, gout, and like conditions may all be present and have to be treated at the same time. Anterior metatarsalgia is a painful complication in many cases, and ofttimes is so annoying to your patient that it is his chief complaint.

The pathology of weak feet that I give you here is quoted from Whitman, whose name is always thought of when this subject is discussed, on account of his extensive studies into this condition. It is, briefly, as follows: "The ligaments in the internal aspect of the foot and of the ankle-joints are weak and distended, the unused portion of the articular surfaces of the joints may be denuded of cartilage, while new facets may be formed to accommodate the changed relations of the bones. For example, the external malleolus may be in direct contact with os calcis; evidences of injury and of abnormal pressure may be found in the thickened periosteum, in formation of osteophytes, while the internal structure of the bones has been changed in adaptation to the new conditions. The disused muscles, particularly the plantar flexors and adductors, have become atrophied, as evidenced by the shrunken calf. The muscles in the inner border of the foot have been overstretched, while those in the outer and upper part have become shortened and contracted in accommodation to the habitual posture.

In discussing the symptoms of weak feet consideration must be given to what the patient can tell you and what you will see. Therefore, you have subjective and objective symptoms. The patient will complain to you of tiring quickly when walking; of pain when walking or after standing for a while; of a feeling of stiffness and difficulty in attempting to walk after a rest; of an indisposition to use the feet as much as formerly. The complaint of pain may include some in the ankles, calves, knee-joints, thigh, hips, and back. This latter, if the patient is a female, may throw you off the track entirely and start you hunting for a gynæcological condition that does not exist. Muscular pain and soreness, with a feeling of strain, are complained of. It is this muscular strain and stretching that is responsible for much of the knee pain that is complained of, and many a puzzling knee condition has been "cleared up" and cured by proper attention to existing weak feet.

If the os calcis tubercles have hypertrophied so that typical exostosis is present, acute heel symptoms of severe pain, tenderness,

and a lack of ability to put weight on the heels will be spoken of. Night cramping of the lower leg muscles is a frequent complaint. Your patient, in many cases, will tell that on arising in the morning an attempt to walk in the bare feet or in slippers will be productive of severe pain, and that every morning his feet are stiff and sore; that this feeling wears off, however, after he puts his shoes on and walks about some. Pathological changes in the tissues ofttimes cause increased pain, stiffness, and disability in damp or changeable weather, which account for the too often wrong diagnosis of rheumatism or arthritis being made. Yet this wrong diagnosis would not, or rather should not, be made if you would examine your patient thoroughly. The objective symptoms are plain and convincing, and ought not to suggest to your mind anything else. Quoting from a previous paper of mine on this subject, published in the *American Journal of Surgery* in April, 1910, they are as follows: Everted feet, the eversion varying in degree anywhere from practically none at all to a very marked deformity; feet with arches partially or entirely "dropped"; feet combining both of the foregoing.

In trying out the normal range of motion of the feet you would find in those cases when stiffness and muscular spasm were present that the motion was limited to a varying degree, this depending naturally on the amount of stiffness and spasm. Any deformity or deviation from the normal outline of the foot could also be seen. If the patient had complained of the heel symptoms, pressure over the tubercle of the os calcis would elicit pain, and also, in some cases, the exostosis can be distinctly felt through the tissues. Pressure along the shaft of the tibia will also give pain. Swelling around the ankle-joint, especially on the outer aspect, is very common, in fleshy patients particularly. Œdema of the tissues of the lower limbs and prominence of the veins may also be apparent, caused by the abnormal posture of the feet when the patient is standing.

I do not know of any condition in which the diagnosis is so very easy and yet where it is so often overlooked and another made. This overlooking of the true state of affairs is by no means monopolized by the younger and inexperienced physician. To many of our older colleagues the condition seems to be absolutely unknown, despite the fact that in this country at least about sixty per cent.

to seventy per cent. of the population are eligible for membership in the "weak feet" club. Many invalids from so-called "chronic rheumatism" are remade, as it were, into value-producing citizens when the orthopædic surgeon is called into consultation by the family doctor.

I take occasion to say right here that it really is surprising to those of us who are interested in orthopædic surgery to see what very little knowledge most practitioners have of this branch of medicine; and how carefully they avoid trying to get any. It is an unfair attitude toward your patients, because conditions coming under the realm of orthopædic surgery arise in almost everybody's practice daily, and only too often pass entirely unrecognized. No one, I believe, can be an expert in every branch of medicine, but we all should try and have a sufficient knowledge of the various branches to enable us to recognize the limitations of our skill and recommend a specialist when one is necessary. It will help to keep down the rapidly-growing number of people who, despite the fact that their financial status enables them to employ a doctor privately, still come to the free clinics for their treatment, because once when they went to a doctor he did them no good and they were benefited at a dispensary. Although in many cases the diagnosis could be made without removing the shoes and stockings, it would be a way of making a diagnosis that would deserve condemnation, for the liability of overlooking some of the conditions would be great. Therefore, have your patient take off both shoes and stockings, even though the complaint is of but one foot, stand erect, and raise the clothes to a mid-tibial height. You may expect to find in this objective examination changes in the feet in keeping with the kind of a case you have before you. In the light case you may find only a slight lowering anteriorly posteriorly of the arch, with very little eversion.

I might mention here that it is my experience that the pain in any case is ofttimes very much out of proportion to the findings, so do not be surprised at small findings in very painful cases. In most of your cases you will find almost obliterated arches and quite some eversion. In others a real flat-foot with eversion. Stiffness and spasm may be present in a varying degree. This, of course, cannot be well demonstrated with the patient standing.

Also, here are two other simple ways of making a diagnosis: Lay a sheet of white paper on the floor. Then spread a thin layer of vaseline on the soles of your patient's feet. Now ask him to stand upon the white sheet of paper and you will get the imprint of the soles of his feet. Study this picture thus presented to you and compare it with the impress of normal feet. How will it differ? In the imprint of the normal foot the middle portion—that is, the part which goes to make up the greater arch or instep—will be represented by a very narrow line. This line will be on the outer side of the foot, of course; you know the arch of the foot is highest at the inner border of the foot and gradually lessens towards the outer aspect of the foot, until it gradually disappears at about the outer sixteenth part of the foot. Therefore, in the imprint of your patient's feet, the widening of this telltale line is in direct proportion to the lowering of the arch. The other way of using this diagnostic method is still simpler. Have your patient step into some water and then walk across the floor. Observe the footprints and diagnose accordingly. Study your patient's shoes. Are they of a shape that would help to ruin the feet? Also stand them on your table and study the shape your patient's feet have forced them into. This malposition will help you make your diagnosis. The wearing out of the soles and the heels along the inner aspect is also a diagnostic point.

You see, therefore, how many things assist you in arriving at a correct diagnosis, but please remember other conditions may exist in conjunction with weak feet, and also there are other things which may be taken for simple weak feet if you are not on your guard. I will mention several of them, with their main symptoms: (*a*) *Acute rheumatism:* An acute onset with pain, fever, and swelling of the involved joints; symptoms involve one or more joints at a time, and are not stationary. There is high temperature; intense sweats of an acid reaction; ofttimes previous history of like attacks: likewise, ofttimes, previous history of disposition to amygdalitis; also a general constitutional reaction. (*b*) *Chronic rheumatism:* Involvement in the symptoms of several joints in various parts of the body; history of previous like attacks, and probably of some acute attack. Pathological changes in several joints; complaints of stiffness, especially in changeable, damp weather. (*c*) *Gonorrhœal*

rheumatism in the acute stage is not so liable to be confused with it as when it is seen in the subacute stage. The history of an acute onset; the history of a past or still present gonorrhœa, the temperature, swelling, pain, redness, and tenderness of the involved part is against simple weak feet. Likewise, examination of the genital organs and the urine also helps to make the proper diagnosis. (*d*) *Tuberculosis of the ankle joint or bones of the tarsus:* This is a very common source of error both ways. However, your history should show pain at night, sensitiveness over affected parts, some lump, white swelling causing a deformity at this site; involvement of only one foot and a lack of desire to bear any weight on the affected side. (*e*) *Syphilis of ankle joint or bones of the tarsus:* History of your case: pain and tenderness in the shafts of the long bones especially, which is worse at night. There is swelling, and should not be very much swelling in deformity. Other symptoms of syphilis elsewhere. (*f*) *Charcot ankle* is comparatively rare, no tenderness; there is a lack of muscular spasm and stiffness. The swelling is mostly due to effusion. There is deformity and there may be signs of locomotor ataxia. (*g*) *Still's disease, arthritis deformans, and other arthritic conditions:* Always more than one joint involved and in various parts of the body. Pain and stiffness may be general, etc. (*h*) *New growths and ankle-joint malformations:* History, appearance, and X-ray picture. (*i*) *Fractures and sprains:* History, swelling, sudden onset, pain, X-ray, etc.

The prognosis in cases of weak feet will be based upon the variety of case you are dealing with, likewise upon duration, occupation, age, general health, and in how well the proper treatment is carried out by both the patient and the doctor. Each must do his share to get the best results. Do not promise every case a permanent cure, but you can promise practically every case relief. You cannot make the ligaments and muscles of the old react as quickly as those of the young; we give them back the strength and elasticity that age has taken from them. Many cases, however, will have to wear their arch supporters only for a time and will then be able to discard them. Others will get the best results by the constant wearing of their foot-plates. By the proper treatment on the part of the doctor I mean that the patient be not sent with either verbal or written direction to a so-called orthopædic

shoemaker to be fitted with weak-ankle or flat-foot shoes or to a bracemaker to purchase a pair of plates. If you are going to attempt to treat without arch supporters and merely with shoes, a treatment I do not believe in as being the best by any means, you must plan your shoe and see that it is constructed properly and fits properly when it is finished. If you are going to treat with shoes in which plates are inserted you must use the same care. It is a very expensive way of treating the cases, the reason why I will speak of later. If you are going to give your patient relief with foot-plates, take the casts for them yourself. Do not rely on others to do the work your patient is paying you to do. If the patient had wanted the shoemaker or the bracemaker to attend to him he would not have come to you in the first place. You must expect a poor result if you give careless or indifferent treatment.

Can we do anything to check the ever-increasing army of weak-footed persons? Men, women, and children crowd our clinics and offices—unfortunately for our prosperity, the former places are the most crowded—looking for cure or, at least, relief from their sufferings and disability. We certainly can do much towards strengthening the feet of the nation. First, the introduction of proper foot gymnastics which will develop and strengthen the ligaments and secondary supporting muscles of the tarsus. Second, by educating our patients to wear proper-shaped shoes; in other words, to purchase shoes that conform to the normal contour of the foot, instead of ruining their feet by endeavoring to force them to conform to the "fashionable" shape of the shoes they buy. Do not stand with your heels together and your toes apart unless you wish to weaken your feet. Stand with your feet in a straight position. Exercise your feet. Standing in one posture for a long time is weakening to both ligaments and muscles, so shift your position occasionally. Don't throw into one foot the weight that Nature intended two feet to support. The stork may be able to do this successfully and without harm to itself, but you cannot. Even those with good feet can conserve their strength by, when sitting or standing, turning their toes inward in the position commonly known as "pigeon-toed." To those whose feet have a tendency to be weak or everted this position is a necessity even when walking. If you try this position you will see that it raises up the arches of

your foot, while eversion causes a lowering of them. Another important factor in prophylaxis is that your patients be kept in good general condition always. Remember that a chain is only as strong as its weakest link and that rust will spread in both directions. This holds good in human organisms also. Constipation is a great factor in weakening the human body. Make your patients develop a daily habit of bowel evacuation.

In the treatment of weak foot we must realize that we have primarily a simple mechanical condition to deal with, or that the mechanical condition may be complicated by some disease. In some cases the removal of the complication and a temporary support to the arches by means of proper arch plates will in time bring about a permanent cure. In all cases we may look for relief. In the simple cases proper arch plates plus massage and feet gymnasties will bring immediate relief and future cure. Some cases will require "stretching" under an anæsthetic, while still a very small minority actual operation. You will see very few of this latter class. Let us take up the treatment of each variety of case separately. I will quote somewhat from a former paper of mine on this subject.

(1) The treatment for simple weak feet, whether the arches are entirely down or not, is first to order the wearing of a shoe which will conform to the contour of a normal pair of feet. These shoes will be shaped so that they will throw the feet into a slightly everted position. The inner border of these shoes will be perfectly straight. There are several such shoes to be had in New York and, I believe, also in other cities. To whatever shoe the patient gets I order added an extra sole about 1/16 to 1/8 inch thick on the edge of the inner border, but gradually decreasing in thickness as it approaches the centre line of the sole, so that when it reaches this point it is practically merged with the regular sole of the shoe. The extra addition to the sole runs from toe to heel, including the instep. This remains in the shoe until the patient is fitted with the proper kind of arch plates. These will be described and exhibited to you in a few minutes. No feet are ready for foot-plates or arches, whichever name you prefer, until the feet are freely movable to overcorrection. Foot gymnastics, strapping, and massage, either hand or vibratory, are ordered. The patient's general condition must be given attention if indications are present for such treatment.

(2) The treatment for weak or flat feet with stiffness and eversion is the same as the above, but, of course, before foot-plates are even thought of, the stiffness must be entirely done away with and instead of fixed eversion you must be able to easily move the feet into an overcorrected inverted position. This is done by manipulation of the foot, putting it through its normal movements. The manipulation can be done by special machine or hand. At each visit of the patient each foot in turn is gradually brought so far as possible without causing too much pain into the positions of flexion and extension alternately for a few minutes. It is then moved to the fullest possible limits of eversion and inversion for several more minutes. Don't try to do too much at a sitting, because the manipulation causes quite some pain, and beyond a reasonable amount at a time your patient will not stand for. After each treatment of this kind the feet are put up in strappings of zinc oxide plaster, in a position of as much inversion as you can get, and a slight amount of flexion. The usual foot gymnastics are ordered to be done at home. When the feet are ready for them the plates are made for the patient. He is discharged from treatment with instructions to report again in from two to three months, so you can see if his foot-plates need to be altered in any way. He is expected to continue his foot exercises. During all this treatment do not forget any complication that exists. It must also be treated. (Figs. 3 and 4.)

(3) The rigid flat-foot which resists both hand and foot manipulation, either because it is too stiff to be manipulated or because the pain caused the patient is too great to be borne, it is necessary to loosen up under an anæsthetic. With the patient under an anæsthetic the feet are loosened up to the extent of being freely movable in all directions, viz., extension, flexion, inversion, and eversion. They are then put up in the position mentioned in the last-mentioned class, plaster of Paris being used instead of the zinc oxide plaster dressing. The plaster-of-Paris dressing is left on from six to eight weeks and the patient allowed to go around. The under surface of the dressing is made heavier than the rest to withstand this walking around, and is flattened to resemble the sole of a shoe. When the plaster dressing is removed the treatment continues as for simple weak foot.

(4) The preliminary treatment in cases of traumatic flat-foot

must be worked out to fit each particular case, bearing in mind the goal you are aiming for, to get to the stage of having the feet in a suitable condition for the fitting of plates.

(5) The paralytic flat-foot also must be studied out carefully. The cause of the paralysis must be taken into consideration. Perhaps a corrective operation must be first performed before a mechanical support would be of service. Perhaps a brace must be used in conjunction with the plates for a time. This must all be worked out by the attending medical man. But remember that no plate should be fitted while any stiffness, spasm, or eversion exists.

Iodides and salicylates do not cure weak or flat feet, but they are useful in conjunction with the regular treatment in controlling some of the pain and stiffness. Do not give them as a matter of routine, but only when they are indicated. Remember, they do not improve the condition of the stomach any. The foot gymnastics I have already mentioned are as follows: (*a*) Instruct the patient to stand "pigeon-toed," then to raise himself up quickly on his toes and come down very slowly until his heels almost, but not quite, touch the floor, then to raise up quickly again on his toes. This should be done every morning and night at least twenty-five times, gradually increasing up to one hundred times. (*b*) Instruct the patient to stand "pigeon-toed" and rise up on the toes, and then come down on the outer border of the feet, gradually getting to the normal position. This also should be done from twenty-five to a hundred times. (*c*) Order your patient to stand in his stocking feet or bare feet and alternately turn his toes upward and downward about fifty to one hundred times. Try these exercises yourself and you will see that they all tend to raise the arches of the feet, besides exercising and strengthening the supporting structures.

The strapping of zinc oxide plaster that I use is different from that used by others. However, I know by experience it is just as efficient and it is by far more economical, and as a minimum amount of plaster comes into contact with the patient's skin it is certainly more comfortable for the patient. It may be described as follows: While the foot is held firmly in a position of flexion and inversion, two strips of zinc oxide plaster, each about an inch and a half wide, are put together, one slightly overlapping the other, and then placed on the foot directly beneath the external malleolus,

Fig. 3.

Weak and flat foot exercising machine for the up and down movement of the foot. The various parts for the different movements are supplied with each machine. The amount of movement is dependent upon the patient who supplies the power to make the machine work.

Fig. 4.

Weak and flat foot exercising machine. This machine exercises the foot in side-to-side movements. The amount of the movement is dependent upon the patient who supplies the power to make the machine work.

Fig. 5.

Position of the foot for the application of the strapping. The two wide straps are put on together, as can be seen in the picture. On the screen just above the patient's leg can be seen the amount of zinc oxide plaster strapping used in the author's style of strapping.

Fig. 6.

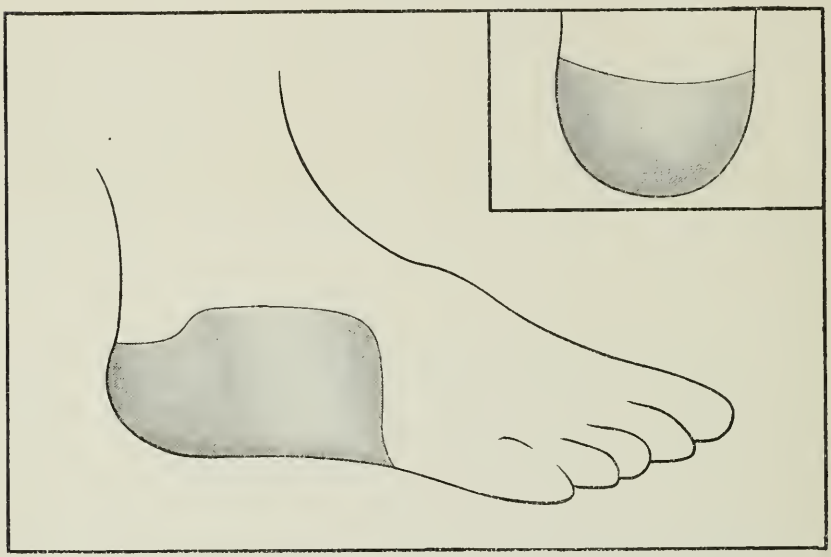
The completed "Steinhardt" method of strapping. In the author's experience it is efficient and economical with a minimum amount of plaster touching the patient's leg. The exact amount of zinc oxide plaster strapping used for each foot can be seen hanging on the screen above the patient's foot.

carried under the sole beneath the arch, and up the inner aspect of the leg to a point somewhere between the middle and upper third of the leg, where it is held in place by a band of zinc oxide plaster applied around the leg. Be careful not to apply this circular band too tightly. Having these two long strips in position, you now start to reinforce this strapping with some narrower bands of strapping previously made ready for the purpose. These bands are about three-quarters of an inch to an inch in width, but of the same length as the original long ones. Still carefully retaining the foot in the corrected position, these reinforcing strips are applied. The first one is applied about an inch to an inch and a half back of the little toe on the dorsum of the foot, about one-half of an inch to three-quarters of an inch above the outer border of the foot. It follows the course of the wide bands under the foot, but after passing around the inner border of the foot it crosses over a little above or at the ankle to about the centre, or slightly past the centre toward the outer aspect, and is made adherent to the circular band holding the broad bands in place. The next band is put on immediately behind and slightly overlapping the first. A few more bands are likewise applied, and you then have the entire arch supported by the broad bands and their reinforcement. I then apply a circular band about a quarter of an inch wide over the original circular strapping to hold the ends of the reinforcing bands in place. As soon as I finish strapping one foot I have the patient put on the sock or stocking. This forces the strapping against the leg and makes it firmly adherent, and, in my opinion, does away with the necessity for the circular band that is used by others in their strapping at the level of the ankle for this very purpose. I usually change these strappings twice a week (Figs. 5 and 6).

Before explaining and also demonstrating to you how to take casts of the feet and prepare them for the bracemaker to make plates from, I want to say a few words as to why I use this treatment in preference to the use of specially-made shoes, either with or without plates built into them. I might mention here that by far the very great majority of orthopædic surgeons the entire country over use the same method of treatment that I do, so you see that it is not an original treatment with me. Whether their reasons are the same as mine I do not know. My first reason for not using the

above treatment alone is that I don't believe in it, because I cannot see how even a properly-shaped shoe is any stronger than an ordinary shoe, and therefore wherein is the already weakened foot to get its support from, not to mention anything about being forced into and held in a corrected position. Or if it is a shoe with a reinforcement in the shape of a leather insert at the instep or with a so-called steel plate built in, there are still the objections that the leather insert is absolutely insufficient to stand the body weight, much less

FIG. 9.



Internal aspect "Steinhardt" plate as it appears in the foot. Such a plate need not weigh over three ounces.

do any corrective work, and gives absolutely in a short time, whereas the steel insert is not only rarely ever high enough to give anything like a proper support to the weakened arch, but is made the same size and height for every pair of feet, whereas not only is every pair of feet different, but most often also is each foot of the same pair. This is a most important reason why they fail to do the work they are supposed to do. I have treated a great number of people in my clinic at the New York Hospital and in my office who before coming to me tried the experiment of wearing a certain shoe of this type that came to their attention through advertisements of it on fences, walls, newspapers, or lay magazines, or circulars through

Fig 7

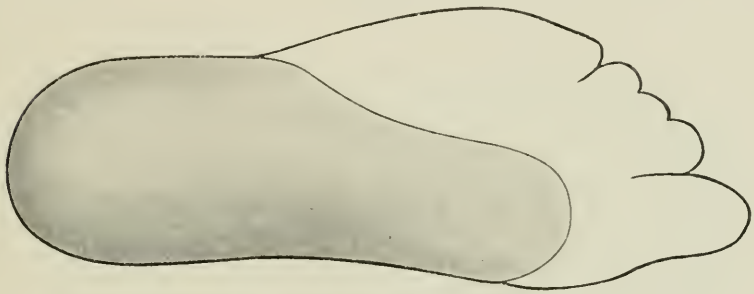
Showing the taking of a cast. Note the position of the foot (which is the right foot) as it rests in the plaster. The patient is sitting well over on his right hip so that he can keep his foot in a position of a right angle, resting in the plaster in its outer border.

Fig. 8.

The completed cast. Note the strips of gauze protruding from between the upper and lower portion of the casts. These strips keep the two portions from adhering together, which allows one to remove them from the patient's foot easily. From these so-called casts the model of the foot is made. The above-mentioned strips of gauze are laid along the upper surface of the lower half of the cast wherever this surface is not taken up by the foot.

the mails. It is even advertised in some medical magazines. Had the shoes of this kind lived up to the claims made for them the wearers would not have required my services. They would have had the relief they sought, even if not a cure. There are numerous shoes of this type, one just about as good as another, in my opinion. The second reason is one of expense. The foot-plates I use are nominal in cost, can be transferred from one pair of shoes to another, and with reasonable care have a life of from two years to two years and a half. I speak now of a good plate in which good material is used. These specially-made shoes are expensive, and even if the

Fig. 10.



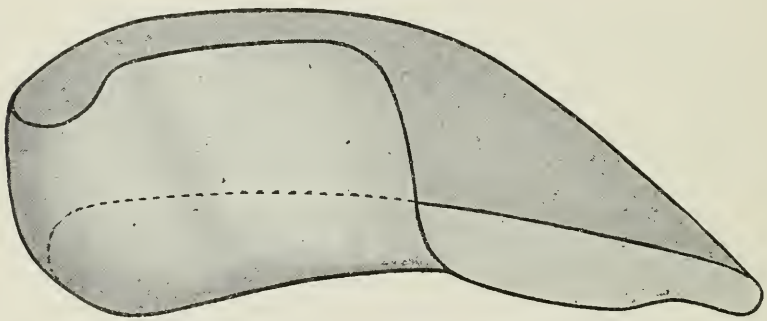
Under side of "Steinhardt" plate fitted to sole of foot.

patient gets along with one pair at a time he will use at least six to nine pairs in the course of two years. No special shoes are needed for the foot-plates. They will fit into any shoes. In clinic practice the question of efficient treatment at minimum cost has to be most carefully considered, and even in private practice cannot be lost sight of. To my mind, therefore, foot-plates are not only the best method of treatment but also the most economical.

To take casts of the feet for plates there is needed plaster of Paris, six strips of gauze bandage, each about one inch wide and twelve inches long, either some vaseline or talcum powder, four ordinary tin wash-basins, four long-handled kitchen spoons, a little common table salt, a piece of non-absorbent cotton batting about three-quarters of an inch to an inch thick and about a foot square, and about four pieces of thin gauze about the same width. You also need a flat-topped stool or box a little bit lower than the seat of the chair your patient is going to sit in (Figs. 7 and 8).

For each foot the procedure is as follows: The patient removes the shoes and stocking and either vaselines the foot from just above the ankle, downward to and including the sole and every part of the leg and foot below the starting point, or rubs it freely all over with talcum powder. Now, supposing it was the right foot you were taking a mould of, he would sit well over on his right hip so that he could easily place his foot in the soft plaster of Paris, with the outer border of it resting flat downward in the plaster. His position must be a comfortable one, for he must hold it without moving the foot until you are entirely through taking the mould of the foot. The stool or box, whichever it is you are using, is placed in front of the patient's chair within reach of the foot, so he can take up the

Fig. 11.

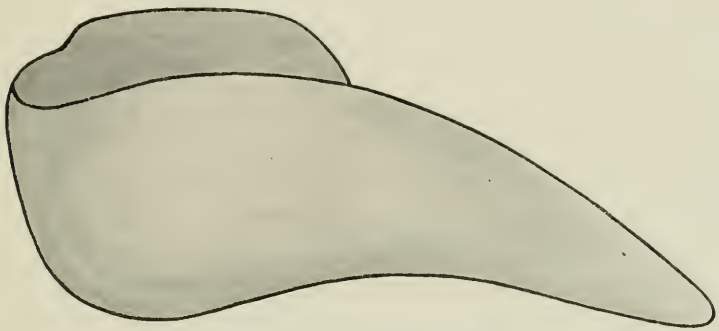


External aspect of "Steinhardt" plate.

position just mentioned when you are ready for him to do so, and on top of it is placed the layer of non-absorbent cotton. Over this comes a piece of gauze or a double thickness of ordinary newspaper. This is to receive the soft plaster when it is prepared. This preparation consists of mixing up some plaster of Paris in some salted water, not stirring it, however, until you see that you have a sufficient amount of plaster in the water for your purpose. Work it until it forms a rather soft, pasty mass. Pour it out on the paper or gauze and smooth it into a layer about half an inch thick, and then place the patient's foot in it in the manner already described. Allow the foot to gently sink into the plaster; do not force it into it. Now mould the plaster rapidly around the lower half of the foot (for it hardens quickly with salt in the water), from the base of the toes to and including the entire heel. Now place the strip of gauze

over the upper surfaces of this half. This will prevent the two parts of the mould from sticking together. Quickly prepare another basin of plaster in the same manner, but with a little more salt in the water, and spread it over the upper half of the foot. Now leave the cast alone until it has hardened or until the patient complains of the heat of the setting plaster. Then, removing the upper half of the cast, take the patient's foot out of the way, put the upper half back into place again, and lay aside. Now repeat this operation for the other foot. You now have an outline of your patient's feet. The next step is to secure the mould of the feet. Take the four halves and vaseline the inner surfaces of each thoroughly. Tie

FIG. 12.



Internal aspect of "Steinhardt" plate.

the half shells from each foot securely together and carefully cover the openings of each pair at the toe ends. Mix up some more plaster of Paris and pour it into these shells through the upper openings, until each shell is entirely filled. Let it harden and then remove the outside shells and you will find you have models of your patient's feet. These you trim out and mark for your bracemaker, who is to follow your markings exactly in making the steel foot-plates, which are to be worn inside of the shoe but over the stocking. The plate I use is one of my own devising, but based on the principles of the well-known Whitman plate, which I formerly used exclusively. You can call this plate of mine the Steinhardt plate or the Steinhardt modification of the Whitman plate. It doesn't matter much to your patient, who will be benefited by it under either name (Figs. 9, 10, 11, 12, and 13).

If the moulds have been well taken they should stand up straight, favoring neither one side nor the other. The highest point on your cast, when laid upside down, should be about two inches from the posterior end of the heel. This corresponds to the anterior portion of the os calcis, which is the point we wish to raise the most. Having marked this point, trim away the plaster from the ball of the big toe backward to this point, and from this point backward to the heel. Always increase the height of the arch so that your plate will overcorrect some. This will give the overstretched ligaments and muscles a chance to contract back to their proper state, and later, when your patient discards the plates, the probable little

Fig. 13

External aspect "Steinhardt" plate as it appears in the foot.

restretching of the arch-supporting structures will probably just be enough to allow the arch to go to normal again. On your now perfected casts map out your plates. Make a mark about half an inch below the external malleolus, next make a mark about half an inch below the internal malleolus, then a mark just behind the ball of the big toe, and finally a mark on the posterior border of the leg, about half an inch lower than the level of the malleoli. Commencing at a point about half an inch behind the external malleolus and about a quarter of an inch below it, run a concave-shaped line backward to the point on the posterior border of the leg, and, continuing from this point, run the line in the same concave shape forward to the point you marked under the internal

malleolus. Now connect with lines the other three points you marked, bearing in mind the following: the external flange of the plates extends from the beginning of the posterior concave line on the external surface upward to the point you marked under the external malleolus and then in a straight line forward to a point directly over and then down to just behind the base of the fifth metatarsal bone; the internal portion of the plate extends backward from the ball of the big toe to the beginning of the posterior curved line on the inner aspect of the foot, gradually rising in a graceful, oval-shaped curve to the point marked below the internal malleolus, when it merges into the posterior curved line. The under portion of the plate will be indicated by a line coming backward from the ball of the big toe to a point at the forward edge of the external flange. It will sort of divide the foot between these points into two triangles. The edge of the plate at the ball of the toe will be rounded. Now blacken all the space enclosed within your lines, and the marking out of your plate is complete. I omit purposely any details of the several cutting operations devised for the cure of this condition. They are very rarely advisable or necessary. This paper is not all that can be said upon this subject, but I hope it has achieved its purpose—to give a working knowledge of this very prevalent condition.

THE INDICATIONS FOR NEPHRECTOMY, WITH ILLUSTRATIVE CASES

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of Boston, Massachusetts

The first statistics of real modern value relative to nephrectomy were published by Küster in 1901. He collected 1,146 operations, of which 287 ended fatally, therefore giving a mortality of 25.04 per cent. I have collected at random 706 nephrectomies performed within the last fifteen years and find 558 recoveries, thus leaving a mortality of 20.9 per cent., an improvement over Küster's report of eleven years ago. Many of the nephrectomies in my collected cases were done for tuberculosis, namely, 155 cases, with a mortality of 40.93 per cent. Such a high death-rate is not surprising, if one reflects that in most of the fatal cases the opposite kidney was found to be diseased or tuberculosis existed in other viscera.

Malignant tumors come next in frequency as an indication for removal of the kidney. I find 116 nephrectomies with a mortality of 25.86 per cent. Now, these malignant growths of the kidney are carcinomata and sarcomata, and when operated on have attained considerable size and have resulted in certain amount of cachexia; therefore, the mortality is in reality not so high as it would at first appear.

The results of nephrectomy for echinococcus of the renal gland are not so good, for out of a total of twenty-six cases I find a mortality of 42.3 per cent.

Injury to the kidney, consisting chiefly of rupture of the renal artery or vein, resulting in such severe hemorrhage that the kidney had to be removed, gives a mortality of only 22.64 per cent. More favorable still is nephrectomy for renal calculi and diseases of the renal pelvis, and I find that the mortality here is 20.5 per cent. Nephrectomy for hydronephrosis gives a mortality of 13.56 per cent., and for pyonephrosis, 10.34 per cent.

The improved results of the operation during the past ten years are due to several precautionary measures introduced. Thus, the general condition of the patient has been considered more carefully

and greater skill has been introduced into narcosis, so that patients with cardiac lesions or cachexia have had a better chance.

Then, too, the nature of the renal affections has been better appreciated, so that the indications for removal of a kidney have become more restricted in many cases. For example, nephrectomy is rarely indicated in nephrolithiasis and never in aseptic cases of renal calculi. Even in infected nephrolithiasis nephrotomy, as a rule, will take the place of the more radical operation, particularly on account of the not infrequent presence of stones in the fellow organ. However, as a secondary operation it has its indications.

The same holds good for cases of severe hematuria which sometimes follows nephrolithotomy and cannot be controlled by other means.

Nephrectomy may become necessary in cases of traumatic rupture of the renal vessels which cannot be sutured, but in many instances control of the hemorrhage may be obtained by ligature or suture.

Hydronephrosis, which also calls for surgical treatment, is not at present considered as justifiable for nephrectomy in all cases, nephrotomy and drainage often being successful. On the other hand, if there is any active parenchyma left, a fistula results which may require a secondary nephrectomy. But the ideal treatment of hydronephrosis is to first discover the cause of obstruction and then, if possible, remove it.

Partial resection of the kidneys for localized growths or abscesses is not to be considered, for the vast majority of cases where this has been done have necessitated nephrectomy later. In some rare instances of localized collections of pus, incision and drainage may be tried, but can hardly be recommended.

Unquestionably, the surest and most important clinical evidence of malignant neoplasm of the kidney is the finding of a mass in the renal region by palpation. By our modern methods of palpation an enlargement of the kidney can be recognized early in the progress of the neoplasm. As hematuria only occurs in about 50 per cent. of cases of malignant disease, its absence in a given case of renal enlargement cannot negative the diagnosis of tumor.

Before deciding on nephrectomy, it is absolutely essential to ascertain if the fellow kidney is present and also to discover the state of its functional activity. For this purpose we have catheterization

of the ureters, and in the majority of cases will give the desired information. To the analysis of the urine from each kidney other tests have been devised which are of great value. The first of these is the phloridzin test. When this drug is given subcutaneously the result is an excretion of sugar which takes place in the kidneys. When both glands are in good functional condition the amount of sugar excreted by each is equal, whereas the urine from a diseased renal gland shows a very distinct diminution in the sugar elimination as compared to the normal kidney.

The second test is by means of methylene blue. Ten cubic centimetres of a 5 per cent. solution are given subcutaneously and the two urines are collected. Under normal conditions the methylene blue appears within the first twenty minutes, coloring the urine blue, and the excretion reaches its maximum within a few hours and should entirely disappear within thirty-six hours. The intensity of excretion is estimated by a color-scale. Under pathological conditions the excretion begins later, continues longer, possibly stops temporarily, and progresses to a lesser degree.

To my mind, however, I think cryoscopy gives the best results. When the kidney secretes the urine from the blood a great osmotic difference in pressure between the two fluids takes place, indicating that the molecular concentration becomes greater in one and less in the other, which can be expressed in figures by determining the freezing point of both liquids. The diminution of the freezing point of a fluid is in direct relation to its molecular concentration. Therefore, the decrease in the freezing point is greater when the fluid is more free of molecules, and *vice versa*. Consequently, when one kidney is healthy it can produce between the blood and the urine a certain osmotic difference in pressure, a difference in the molecular concentration which can be expressed in figures, while we determine the diminution in the freezing point of the liquid, which is, under normal conditions, in relation to the blood $\delta = 0,56°$, and to the urine $\Delta = 1.2°—2.2°$. If pathologic changes have taken place in a kidney, then the osmotic difference in pressure between the blood and urine does not occur; the molecules remain in the blood—a fact made evident by a greater decrease in the freezing point of the blood and that of the urine lesser. The blood is taken from a vein, but later oxygen must be introduced in order to expel the carbonic acid contained,

because it causes a rise in the freezing point. If the freezing point is below 0.58° nephrectomy may be considered unsafe and is a good working rule.

But of far greater import is the twenty-four hour amount of urea, and upon this mainly rests the indication of interference.

Although many have decried these various tests, it appears to me that their arguments are very largely theoretical, but when all is said, the proof of the pudding is in the eating, and in many cases clinical observation and objective demonstration, aided by these tests, combined with the results of experience, suffice in the determination of a correct diagnosis.

To bear out this statement and as illustrative examples of nephrectomy, the following cases will be reported:

Case I.—Female, æt. 41 years. At the age of twelve had typhoid fever; otherwise had always been in good health. Married at the age of 23, has had three children, all of whom are alive and well. Present illness began four years ago with pain in right lumbar region. For three years this has increased, and at about this time the patient noticed an enlargement of the right half of the abdomen. Nine months ago frequency and pain at micturition commenced, and the abdominal enlargement was more noticeable. The dysuria is now very marked.

Examination: Small, poorly-nourished woman. Heart and lungs normal. Right half of abdomen distinctly protrudes. Circumference on right side 44 centimetres, 35 centimetres on left. Tumor reaches from costal border to pelvis and extends to median line. Posteriorly it becomes lost in the lumbar region. Percussion is dull all over the growth, but a tympanitic area exists between it and the liver. The surface is smooth and the tumor cannot be moved. Evident fluctuation as well as tenderness on pressure. Amount of 24-hour urine varies from 600 to 1000 cubic centimetres. Urinalysis negative. Micturition about every hour and a half. No elevation of temperature, pulse 78 to 80 per minute.

The diagnosis of a closed pyonephrosis was made, and since the amount of urine was diminished, but normal in composition, it was evident, without further examination, that the opposite kidney must be functionally normal. Therefore, no hesitation could be entertained as to the performance of nephrectomy, which was done two

days later. The patient was placed upon the left side and the incision carried from the twelfth rib forward to the anterior superior spine of the ilium, including skin and muscles down to the peritoneum. The serosa was stripped off the enlarged kidney and after careful gauze packing the organ was punctured, giving issue to about 490 cubic centimetres of thin, alkaline-smelling pus. After emptying, the sac collapsed, and it was then easy to free the kidney to the hilum. The ureter was tied off separately, and then the vessels.

There was little shock after the operation, and the patient was discharged in good condition three weeks later. Examination of the specimen showed a flat layer of renal parenchyma lying on the sac. The patient died six years later from pulmonary tuberculosis.

Case II.—Female, æt. 31 years, negative family history. Married when 26 years of age. History of gonorrhœa in the husband six months before marriage. First and only confinement three and a half years after marriage. Pregnancy and delivery normal, but after the patient was up she noticed that the urine was cloudy, and complained of much headache. Several months later a large amount of albumin was discovered, and the patient was placed under treatment. For some time she improved, but again relapsed, this time becoming so weak and anæmic that she was kept in bed. I am unable to discover what the diagnosis was at the time, but a nephrotomy was done by a surgeon in one of the smaller cities of the West. Three months after this she came East and consulted the writer.

Examination: A tall, well-built woman, very anæmic, hæmoglobin 47 per cent. Heart and lungs normal. A renal fistula in left lumbar region giving exit to cloudy urine, containing many pus-corpuscles but no renal cells. Cystoscopic examination showed considerable hyperæmia around the left ureteral orifice. No renal cells in bladder urine, but albumin one gramme per litre by Esbach's tube.

The patient was seen five days later, and examination of bladder urine revealed no albumin. The patient was kept under observation for two weeks, during which time the albumin and a few pus-cells would appear in the bladder urine, and from this it was assumed secretion from the left kidney was at times interrupted. There was a slight evening rise of temperature of a degree or two.

A diagnosis of pyonephrosis was made and nephrectomy advised and performed a few days later. An incision was made in the old scar

of the nephrotomy, but carried farther downward and anteriorly. Much difficulty was experienced in exposing the kidney, as the organ was firmly adherent to the fatty capsule, but by careful dissection it was finally freed. The kidney was enlarged and soft and ruptured as it was being brought out of the wound, giving issue to some thick pus. The pedicle was ligated and the organ removed. The wound was packed with gauze and the incision partially closed.

The patient made a good convalescence and returned to her home in the West two months later. Fourteen months afterward she was delivered of a healthy baby, both pregnancy and labor being normal. Her health has continued excellent and the headache and pain in the renal region have disappeared.

Examination of the kidney revealed numerous abscesses corresponding to the renal calyces. The pus was sterile. Very little parenchyma remained.

Case III.—Female, æt. 64 years. Family history negative. Two children, both alive. In August, 1905, patient noticed a tumor in the right lumbar region, which slowly enlarged without giving rise to any symptoms. She was seen the following September.

Examination: Small, thin woman. Thoracic viscera normal. In the space between the costal border and the iliac crest on the right a prominent tumor can be palpated. It is hard, elastic, and can be moved inward and upward, especially backward. Its size was estimated to be that of an orange. No tenderness on pressure. Urine normal in quality and quantity. A diagnosis of renal cyst was made.

Nephrectomy was done a few days later. The kidney was exposed with ease, as the patient was very thin. The lower pole was involved by the globular tumor felt by palpation. The surface was smooth and adhered to the peritoneum, part of which was removed with the kidney and the opening closed. The vascular mass was tied off, the cavity packed with gauze, and the incision partially closed. Recovery uneventful.

The specimen showed that the tumor was quite round, having a circumference of 12 centimetres, with a distinctly developed capsule. On section it presents a uniform grayish-red color. Microscopically it proved to be a small round-cell sarcoma. In May, 1907, the patient began to notice an increase in the size of the abdomen (ascites), with marked constipation. The right lumbar region

appeared normal, but in the left hypogastric area a tumor the size of a man's fist could be felt. Exploratory incision was not considered advisable, and she died three months later. No autopsy could be obtained.

Case IV.—Male, two years and two months old. Parents healthy. One other child, four years old, healthy. For about four months the mother had noticed an abdominal tumor which had developed rapidly. Blood had never been noticed in the urine, and the child's general health did not seem to suffer.

Examination: Well-nourished boy, but rather anæmic. Otherwise he appeared well. The right half of the abdomen prominent, and here a firm, elastic tumor could be palpated, extending from just below the costal border to the inguinal region. It reached about one finger's breadth over the median line. Percussion and palpation show the tumor fills the right side. It has a slight lateral mobility. Bimanual palpation failed to detect it posteriorly, while percussion showed a tympanitic area between the tumor and the liver. Urine normal.

Given the history and location of the tumor, a diagnosis of right kidney neoplasm was made, and, since the urine was normal, it was assumed that the opposite kidney was functionally good. On account of the large size of the growth nephrectomy was done by the abdomen. The peritoneum covering the anterior aspect of the tumor was split and the growth carefully dissected out, but it ruptured, allowing the escape of soft neoplastic tissue. The pedicle was tied off and the posterior peritoneal incision closed. The latter had been made about 2 centimetres internally to the ascending colon. The abdominal incision was closed in layers.

The child was in profound shock when he left the table, but by careful nursing he rallied. He made a good operative recovery, but died about seven months later from recurrence of the growth.

The tumor retained the shape of the kidney, the lower two-thirds being much more developed than the upper. Near the lower pole a small rent is found which enters into a capsulated cavity containing a soft tumor mass. The tumor weighed 1,075 grammes and measured 15 centimetres. On longitudinal section three growths, apparently similar in nature, are seen. The larger, which is about the size of a closed fist, occupies the lower two-thirds of the kidney. Of the two smaller ones, the one lying directly at the upper pole is the size

of a walnut, the one below it is about the volume of a chestnut. A narrow strip of cortex exists, becoming very thin over the larger tumor.

Microscopically, the growth shows quite a uniform structure. Groups of cells, some round, others oblong, in places connected together, are surrounded by a loose tissue, poor in cells. Epithelial elements of glandular character are seen in the centre of most of these cell groups. The epithelium, with its elongated nuclei, is sharply defined against the surrounding tissue, rich in nuclei. These cell masses around the epithelial cells consist of spindle-shaped nuclei without any evidence of protoplasm. The border line of the interposing tissue is well marked, and the cell masses are so evident from their dark pigmentation that a kind of alveolar structure results. This framework, with its scanty cells and fibrous elements, contains spindle-shaped nuclei arranged in rows around the cell groups. Stellate-shaped cells can only be distinctly seen here and there, but the growth as a whole appears to be very loosely composed, like mucous tissue. With thionin it stains a Burgundy red. Where the neoplasm joins what remains of the renal parenchyma it appears to have pushed it aside, and the line of demarcation is well defined. The pathological diagnosis is, therefore, malignant adenomyxosarcoma.

Let us consider these four cases a little more in detail relative to certain points they present. In the etiology of Case I there is a suspicion that the pyonephrosis was tuberculous. However, there was nothing in the patient's hereditary antecedents, and the general physical condition gave no evidence of tuberculosis. However, as she ultimately died of pulmonary phthisis, it is probable that the renal lesion was due to Koch's bacillus.

In Case II, although obscure, the renal infection was probably from the gonococcus. The husband contracted the disease six months before his marriage, and although there is no evidence of acute infection of the wife, still the habits of Neisser's organism are so varied that I infer it was the etiological factor in this case. I am decidedly of Isreal's opinion, namely, that most pyonephroses in otherwise healthy individuals owe their origin to the gonococcus, although one must never forget the intestinal bacteria, particularly the colon bacillus.

From the clinical course of these cases it may be assumed that in

Case I the functions of the diseased right kidney had completely ceased; probably a displacement of the ureter had taken place which blocked the flow of urine to the bladder and resulted in a complete obstruction. Atrophy from back pressure then resulted, with loss of physiological function of the parenchyma. Only the urine from the left kidney reached the bladder, and, as it was normal, one could infer that the kidney was functionally good. One was, consequently, dealing with an encapsulated pyonephrosis.

In the second case plugging of the ureter was intermittent, so that the urine would from time to time be free from pus and albumin. The ureteral obstruction in this case was undoubtedly due to thick plugs of pus.

In both the cases of sarcoma it is interesting to note that at no time was blood found in the urine. However, in only about 50 per cent. of malignant renal disease does hematuria occur, so that when in the presence of a growth, apparently of renal origin, *without* hematuria, it should not cause one to reject the diagnosis of a malignant renal neoplasm.

In Case I the differential diagnosis to be considered was hydronephrosis, and the normal composition of the urine might at first lead one to suspect this condition, but the severe dysuria was more in favor of a pyonephrosis. In Case II a pyonephrosis was made evident by the nephrotomy. In the two last cases a fairly correct diagnosis could be made. By palpation in the case of the woman it was evident that the growth was quite movable, particularly backward and upward toward the lumbar region, so that a movable kidney undergoing malignant transformation seemed evident. A renal cyst was also to be considered in the differential diagnosis, particularly so on account of the pseudofluctuating consistency of the growth.

In the case of the child it is interesting to recall that the tumor could not be palpated in the lumbar region bimanually, and differentially the diagnosis of an omental growth or of the mesentery could not be excluded, but the very great rarity of these tumors in childhood made malignant disease of the kidney seem more likely. From the view-point of pathology the tumor was interesting from the fact that it proved to be an embryonal adenosarcoma, probably developing from hyperplasia of fœtal tissue, the remains of the wolffian body.

A CASE OF STAB-WOUND OF THE HEART: TRANS-DIAPHRAGMATIC PERICARDIOTOMY: RECOVERY

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A HEALTHY boy aged twelve years was assisting in the unloading of a cart of hay when the horse moved and he fell from the top of the load together with a three-pronged hay-fork. The hay-fork struck the ground handle first and the lad fell on one of the prongs, which penetrated the upper part of the abdomen just outside the left rectus muscle, an inch below the free border of the ribs. Without loss of time he was conveyed, a distance of some three miles, to the Dundee Royal Infirmary and on admission was obviously in a serious condition. He lay on his right side with the spine flexed and the knees drawn up over the abdomen. He resented examination but complained of pain in the region of the stab. He wore an anxious expression and was extremely restless. There was some retraction of the abdomen and the abdominal muscles were hard and rigid. The face was of good color, his breathing was thoracic and his alæ nasi were active. There was slight dyspnœa, no cyanosis. The pulse was 80, regular and of fairly good quality. The heart's area of dulness appeared to be diminished. There was no sign of pneumothorax nor hæmothorax. The heart sounds were audible and apparently normal.

The boy's condition precluded a more thorough investigation, but the position of the wound, the rigidity of the abdomen and his obviously serious condition suggested rather an injury to an abdominal than to a thoracic viscus. Chloroform anæsthesia was induced, the abdomen opened in the middle line above the umbilicus.

It is my experience that in cases of stab or gunshot wound of the abdominal parietes, it is better to open in the middle line than to make an incision in any way related to the track of the weapon. A sufficiently large opening can thus be made and the abdominal wall on the injured side so raised that its surface may be inspected, when injury or soiling of the peritoneal surface cannot escape notice. A fairly common abdominal injury of this nature in civil practice, is that caused by a bullet from a pistol of small calibre with a small charge and low muzzle velocity. The penetration power from such a firearm is very indifferent and so I adopt this plan of examination in every case, whether it is uncertain or not that the bullet has penetrated, though it is known to have entered, the abdominal wall.

This method of examination being carried out, on raising the left side of the abdominal wall, it was seen at a glance that the peritoneal surface had been injured. Particular examination showed that the prong which had caused the wound had passed very obliquely upwards in front of the left lobe of the liver but clear of it, leaving it uninjured. In this neighborhood there was a

little blood, and on clearing this away a puncture was discovered in the diaphragm. On a director passed through the puncture the tendon of the diaphragm was slit a little in both directions and so the pericardial sac opened. The pericardium contained a considerable quantity of blood, fluid and clotted, and this was removed by swabbing and the removal was followed by distinct relief. There was then observed a punctured wound on the left ventricle of the heart fully an inch from the apex. This wound was plugged with a small blood-clot and there was no oozing from it. The heart was freely handled and there would have been no difficulty in inserting sutures had these been called for. The wound in the diaphragm was closed with interrupted catgut sutures, and the abdominal wound in the usual way.

On the following day the heart's dulness was present, temperature 100.4 ° F., pulse 120, and slightly irregular; the patient retained his anxious expression, the breathing continued somewhat labored, and he was still inclined to be restless. On auscultation the heart sounds were distinct and normal, but over the third left costal cartilage, and in the third intercostal space anteriorly, loud pericardial friction could be heard. All symptoms of urgency disappeared during the succeeding twenty-four hours, the pulse became regular, the heart's dulness normal, the anxiety and breathlessness disappeared, but a slight restlessness still continued. A small stitch abscess gave a little discomfort during the next day or two, but it was healed by the fifteenth day, by which time all discomfort had vanished, no pericardiac friction could be heard, and the pulse was 88, quite regular, and normal in rhythm and force. His further convalescence was entirely uneventful, and he has now remained perfectly well and able to work, as a farm servant, during five years.

In the above case there are two interesting points for consideration: the wound of the heart and the wound of the diaphragm.

Much has been written about wounds of the heart, and as no individual experience can be very great, many single cases have been reported; while every now and again a writer has gathered these together and tendered generalizations and deductions made from the larger number. One of the most recent papers on the subject comes from the pen of Mr. Bland Sutton.[1] The interest and danger of the condition naturally increases with the size and depth of the wound. My case cannot compare in severity with some of the cases published. I cannot even say whether the wound reached the cavity of the heart or merely punctured its wall, but in any case the wound was a small one, it seemed efficiently plugged with clot, and though suturing could have been easily carried out, it was not called for.

The danger of the injury, apart from immediate death from shock, lies in the effusion of blood into the pericardial sac and the interference on this account with the heart's action. It is for this, hæmopericardium, that early operation is called for, and suturing of the heart-wall is undertaken merely as an accessory. I do not

maintain that from the experience of one case a transdiaphragmatic route is the best for pericardiotomy, but I have had occasion to open the pericardium in several cases for other than traumatic conditions and by no other route did I obtain the same freedom of examination of the heart nor ease of handling it. Doubtless the ease of examination and of manipulation will be materially contributed to by the shape of the chest and the resiliency of the ribs and in youth such conditions generally obtain. Pericardiotomy is usually carried out for inflammatory conditions with effusion of serum or pus, and access is gained to the sac by removal of certain costal cartilages. It can easily be done without wound of the pleura, but my impression from several such cases is, that though the sac is easily got at and cleared, manipulation of the heart would be somewhat difficult. On more than one occasion I have removed for tuberculous disease several costal cartilages on both sides, and part of the sternum. This exposes the anterior surface of the pericardium freely and would give good access to the heart, but it is a considerable operation, much more severe and occupying much more time than transdiaphragmatic pericardiotomy, which latter presupposes an uninfected pericardium and its practicability depends on the ultimate result to the wound of the diaphragm.

From experience gained just one hundred years ago Guthrie,[2] when he wrote his "Commentaries," stated, "A wound of the diaphragm never closes except under rare and particular circumstances; but it remains open during the rest of the life of the sufferer, ready at all times to give rise to a hernia, which may become strangulated and destroy the patient unless removed by an operation as yet unperformed, but to which attention is specially directed." He gives, more or less in detail, several most interesting cases of wound of the diaphragm, and adds, "These cases confirm the fact I was first to point out . . . that wounds of the diaphragm, whether in the muscular or tendinous part, never unite, but remain with their edges separated, ready for the transmission between them of any of the loose viscera of the abdomen which may receive an impulse in that direction. That parts of these viscera do pass upwards and back again cannot be doubted; and it is probable that incarceration may take place for a length of time before strangulation occurs from some sudden and distending impulse giving rise to it."[3] It can scarcely be doubted that several cases can now be recorded in which "under rare and

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particular circumstances" a wound in the diaphragm has healed. It would be interesting to have the result of a post-mortem examination where a wound of the diaphragm had successfully healed, but I am unacquainted with any such observation. These "rare and particular circumstances" which Guthrie mentions probably consist nowadays in the promptness of efficient attention rendered to the patient, his conveyance without any exertion on his part, and the surroundings to which he is brought in any modern hospital. One is so accustomed to associate defects in the diaphragm, whether traumatic or congenital, with herniated viscera, and this herniation with strangulation in traumatic cases, that Guthrie's extraordinary case of Sergeant Barry is worth referring to.[4] Sergeant Barry received a gunshot wound on the day before the battle of Fuentes d'Onor in 1811, the bullet entering close to the left nipple in front and passing out between the eighth or ninth rib posteriorly. After various portions of his clothing had been discharged from the wound, he improved rapidly and rejoined his regiment. He died in 1833, from "mortification of the left leg." On postmortem "the whole of the stomach and the greater part of the transverse arch of the colon were found in the left cavity of the chest, having passed through an opening in the diaphragm, extending about three inches in the transverse direction, near the centre of the dorsal attachments of that muscle." The tendinous part of the diaphragm was not affected. With a known wound of the diaphragm and a suspected hernia of viscera into the chest Guthrie gives certain recommendations, but he writes, "If symptoms of strangulation should come on, an opening made in the left abdomen would appear to offer the only chance of life."

Ballingall,[5] another old writer on military surgery and a contemporary of Guthrie, gives but small space to wounds of the diaphragm and has apparently no personal observations to record. The cases he offers are not at all unlike those of Guthrie, but he is a more conservative surgeon possibly, for he does not suggest surgical interference. Probably this dictum of the greatest authority of his day has rightly influenced subsequent opinions on injuries of the diaphragm, and in this country, where brawling is rarely accompanied by the use of the knife, stab-wounds of the diaphragm are more uncommon than they appear to be in Italy and France. From these countries admirable monographs on the subject have emanated.

But though surgery has been revolutionized since the Peninsular War, Guthrie's pronouncement still merits respect. Paget,[6] in one of the most recent English works, writes, "From its (the diaphragm's) constant movement, it fails to heal where it has been wounded or the scar is weak and may break down years afterwards."

Litler Jones[7] writes, "It is well to remember that wounds of the diaphragm through the tendinous portion may subsequently yield or heal indifferently." He gives a case in which he successfully operated for a stab-wound in the diaphragm not involving the heart. The wound was partly muscular and partly tendinous and was sutured by five interrupted sutures of chromicized catgut. He records another case on which he operated, a girl aged 8 years, who developed a strangulated diaphragmatic hernia of the left side eight months after an abdominal injury which, the author thinks, was sufficient to cause a rupture of the diaphragm. The operation was not successful and a good deal of difficulty was experienced in reducing the hernia. As regards the surgical treatment of the hernia through the diaphragm, he writes, "The general preference is for the abdominal route or the combined" (thoracic and abdominal). In order to effect reduction of the hernia in Jones's second case, he had to enlarge the opening by slitting on a director.

Paget[6] quotes two cases from Frey[10] where an abdominal wound of the diaphragm was successfully sutured, one primarily, and the other secondarily, on account of a diaphragmatic hernia following the injury—this hernia contained colon and omentum. He states that Frey collected 33 cases of wounds of the diaphragm, and of these 88 per cent. ended sooner or later in death. In only seven cases out of twenty-one who died from strangulated diaphragmatic hernia did the event occur at once or soon after the injury, and in fourteen it occurred within a year after apparent complete recovery, though in some, warnings of the impending disaster were not wanting. Later he writes,[11] "The best way to reach a diaphragmatic hernia is through the pleura, not through the abdomen." The urgency of wounds of the diaphragm apart from the immediate shock or bleeding seems to depend chiefly on the herniation of some viscera from the abdomen into the chest.

The punctured nature of the wound in my case obviated such a complication as hernia. It seems, too, in accordance with experience in other movable parts, that wounds of the diaphragm, recent and

uncomplicated, or made during an operation, should heal and the wound become properly cicatrized. The protection from the liver on the right side, or the seriousness of a wound of that organ makes it natural that wounds in which the diaphragm requires treatment should be more commonly found on the left side than on the right. The freedom from injury of the abdominal viscera has been commented on, and it is supposed that the weapon having lost its force rather pushed the viscera aside than perforated them, but it must always be remembered that most of these wounds are in cases of attempted murder or suicide.

The direction of the wound must be taken into account. If the wound passes through a lower intercostal space more or less at right angles to the surface of the chest, it is possible that the pleura may escape injury while some abdominal viscus may meet with harm. If the wound is from below upwards as in my case, the certainty is that the abdomen is punctured, the uncertainty being what has happened to the diaphragm or to the thoracic viscera above. If the wound is directed from above downwards and is followed by a pneumothorax or with pericardial effusion one has the certainty of knowing the thoracic injury, but if it be a diaphragmatic one the uncertainty is as to the condition of the viscera below.

Sir William MacEwen [12] has pointed out that the admission of air to the pleural cavity does not mean that absolute collapse of the lung which was formerly taken for granted. This is entirely in accord with my experience, provided that the opening be large enough so that the air sucked into the pleura can get out again and is, therefore, not accumulated under pressure. In a wound of the lung this is apt to occur and thoracotomy will give relief, and it is for this reason that a wound of the lung is not followed by immediate marked pneumothorax unless the injured person exerts himself as in struggling, running, or shouting for help. In any case except where serious pneumothorax made immediate thoracotomy imperative, it seems that it might be advisable to do a median cœliotomy for examination of the abdominal contents and inspection of the diaphragm, before interfering with the thorax. It stands to reason that the abdominal viscera must be more easily handled and more thoroughly observed through an abdominal incision than they can be when any attempt is made to examine the abdominal contents transthoracically, though it must be admitted that reduction of a

hernia is safer by compression from above the diaphragm than by traction from below. Nearly all the writers on the subject, however, recommend the transthoracic route and Bucci [13] appears to have operated thus on two occasions, suturing the stomach in one case and the spleen in another, through the thoracic incision aided by separation of the ribs with costal resection. It is not easy to realize how far mere forcible separation of the ribs can be carried out. Sauerbruch and Schumacher [8] figure a powerful instrument for the purpose. If the reality is anything like their illustrations there must be a fair amount of room obtained. The younger the patient in whom the diaphragm is inspected through the abdominal incision the better, for the flexible ribs can be more easily raised and pulled out of the way than in old people where the ossification is more complete.

Few will be found to differ from Schwartz [9] that the proper course is immediate operation. Abstention gives a mortality of 90 per cent., but no decision as to whether the thoracic, or the abdominal or the combined route be the best can be given, for the route adopted must depend on each particular case, and its possible complications.

REFERENCES

[1] Bland-Sutton, J.: Treatment of Injuries of the Heart. *Brit. Med. Journ.*, Lond., May 28, 1910.

[2] Guthrie, G. J.: Commentaries on the Surgery of the War. 5th Edition. Lond., 1853, 477.

[3] Guthrie, G. J.: *Op. Cit.*, 481.

[4] Guthrie, G. J.: *Op. Cit.*, 479.

[5] Ballingall, Sir G.: Outlines of Military Surgery. 4th Edition, Edin., 1852, 322.

[6] Paget, S.: Surgery of the Chest. Bristol, 1896, 140.

[7] Jones, T. C. Litler: Hernia of the Diaphragm. *Liverpool Med. Chirurg. Journ.*, Liverpool, July, 1909, 359.

[8] Sauerbruch, F., und Schumacher, E. D.: Technik der Thoraxchirurgie. Berlin. 1911, 23.

[9] Schwartz, A.: Traitment des Plaies Thoraco-abdominales. *Paris Médicale*, No. 8, January 20, 1912, 194.

[10] Frey: Zur Casuistik der Zwerchfellsverletzungen. *Wien. med. Wochenschr.*, 1893, 160.

[11] Paget, S.: *Op. Cit.*, 151.

[12] MacEwen, Sir W.: Some Points in the Surgery of the Lung. *Brit. Med. Journ.*, July 7, 1906.

[13] Bucci, F. P.: Contributo alla cura delle ferite toraco-diaframmatiche. *Clin. Chir.*, Milano, 1908, vol. 16, 920–966.

[14] Eppinger, H.: Allgemeine und Spezielle Pathologie des Zwerchfells. Wien, 1911.

WHEREIN THE DIAGNOSIS OF PELLAGRA IS OF SURGICAL IMPORTANCE

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The medical profession of America owes a very distinct and at the same time a great debt of gratitude to Dr. J. W. Babcock, of Columbia, South Carolina, for first calling attention to the existence of pellagra in this country; he not only called attention to the malady, but described it, named it, recognized it, and proved its existence. Until a short while ago this disease was only known as a curious name printed in some of our text-books; now we know that the disease has existed in this country for nearly one hundred years; we know that in some sections, especially in the South, the number of cases has reached almost epidemic proportions. The condition is of such importance that both the State and Federal governments, in many instances, are working together in an effort to solve the riddle of its cause. Before I can be justified in attaching surgical importance to such a subject as this (generally thought of as entirely a medical malady), two things must be proven. First: that pellagra is of general interest. In support of this first proposition, we submit the following: that pellagra is of interest to medical men living in States other than the strictly Southern States is proven by the fact that cases have been reported from, and are known to exist in, about thirty-eight States of the Union: for example, Massachusetts, New York, New Jersey, Pennsylvania (at the Philadelphia Hospital for the Insane, Blockley, a very interesting focus of infection has been discovered), Kentucky, Iowa, Kansas, California, Ohio, New Mexico, and Colorado. At the present time there are at least three hundred cases in the State of Illinois.

Dr. C. H. Lavinder, one of the government men detailed to investigate this problem, says that twenty-five thousand is a very low estimate to place on the number of cases existing in America to-day. To make a long story short, pellagra exists from the Atlantic to the Pacific and from Canada to the Gulf of Mexico. This is no

careless statement, but an accurate one entirely susceptible to demonstration and proof; a disease, therefore, so widespread must surely be of general interest to the medical profession. We feel safe, then, in announcing the general proposition that pellagra is of such interest, and leave it with you to determine whether or not this part of my case has been made out.

Second: it is now incumbent upon me to show, surgically, that there are cases, quite a number of them, both acute and chronic, that can be, and are, mistaken for surgical conditions.

Pathology.—This is from an autopsy report of one of Lombroso's cases. Œdema malleoli and decubitus. Cerebral convolutions slightly atropic; floor of fourth ventricle anæmic; heart and aorta slightly dilated; aortic segment thick; lungs œdematous; spleen and liver congested, both had spots of fatty degeneration. Kidneys soft; cortical substance very thin. Bowels atropic; muscular coat and intestinal mucosa very anæmic; hypertrophy of the salivary gland, mesenteric glands reddened.

Spinal Cord.—The inside dura appeared to be polished; the pia was thick and opaque, especially on the dorsal surface; on the arachnoid were many hard, small placques that resembled bone. Meninges were hyperæmic. The consistency of the cord was decreased in the cervical region; the gray matter resembled (one section) commencing decomposition. Microscopical examination of the cord showed degeneration of the pyramidal tract. This lesion was symmetrical from the site of the fifth pair of cervical nerves and increased as it descended the cord, with maximum at the lumbar vertebræ. On this pair very few nerve fibres were preserved, the nerve fibres being replaced by sclerosed nerve tissue. The posterior columns were very slightly affected and limited to the column of Goll and a few fibres of the column of Burdach. This degeneration stopped at the first cervical nerves, where the sclerosed nerve tissue could not be seen. The ganglion-cells of the gray matter, especially on the anterior cornu of the dorsal region, were atropic, very pigmented. The principal pathological lesion was in the spinal cord, and the pyramidal tract had suffered most, next to the ganglia as well as the anterior cornu.

Symptoms.—I shall give a very brief description of the symptoms and refer those interested in this subject to a splendid article by Dr. Watson on the Symptomatology of Pellagra, published in the

International Clinics, Volume I, Series 20. The symptoms here given are culled almost entirely from this paper.

Digestive Disturbances.—"The buccal mucosa becomes very red, the tongue and mucosa of the lips showing particularly this characteristic symptom. If the throat is examined this redness will be observed as far as one can see into the pharynx. Flakes of exfoliated epithelium will be seen adhering to the gums, and the tongue, being denuded of its epithelium, is smooth and glistening. Its color is a cardinal-red, and it is an important symptom of the disease. I have denominated it the cardinal tongue."

Stomach.—Stomach symptoms may at times be most pronounced. "The acme of the stomatitis corresponds in point of time and severity to the acme of the eruption on the hands. Other mucous membranes are also inflamed, protoscopic examination revealing a bright red mucosa as far up the gut as can be seen." [2]

There are also gastric crises with occasional hemorrhage.

Diarrhœa.—Diarrhœa is almost always present during some part of the disease, varying in intensity and degree; sometimes the stools contain both blood and mucus.

Pupil Symptoms.—Pupil symptoms are at times very marked and pronounced in their variations, due probably to the involvement of the cilio-spinal centre, which is situated in the cord between the first and second dorsal vertebræ, a part of the cord always involved in the disease.

Temperature and Pulse.—Temperature and pulse depend upon the severity of the infection; high temperature and rapid pulse belonging to most malignant and fatal infections.

Psychic Phenomena.—"Briefly, mental depression is as constant as the erythema and diarrhœa, and varies from a mild case of the 'blues' to severe melancholia." [3]

Skin Eruption.—The skin eruption is characteristic of the disease; the only trouble about the eruption is that it is frequently a late symptom and occasionally does not occur. If it were a constant and real symptom, mistakes in diagnosis could hardly happen. It is an erythematous rash, closely resembling sunburn and occurring on the back of the hands—parts exposed to the sun.

[2] Watson.

[3] Watson.

So much, then, for the symptoms. Now to our real undertaking. A malady with the symptom-complex of digestive disturbances, stomach symptoms, diarrhœa, psychic phenomena, etc., can, in the atypical case, be mistaken for anything. We know, for example, that the poison of pellagra is especially liable, in women, to involve the female pelvic organs. "In the early cases we have all the train of symptoms: backache, pelvic distress, headache, bearing-down pains, weakness, lassitude, emaciation, ovarian and menstrual disturbances, which are seen in pelvic disease, for, as Manton well says, 'mental symptoms are present in gynæcological cases.'"

When the eruption is not present, the unsuspecting doctor or consultant is most likely to pronounce the case pelvic, and treat the symptoms as pointing to a primary gynæcological trouble, without thinking of its being a part of the pellagrous symptomatology. Here, too, the mental depression is regarded as merely a symptom of the pelvic disease, and operation or special treatment is advised. The consultant neglects to ask for a history of the eruption for one year, or maybe for four or five years, and at the same time there is perhaps a very red tongue, swollen and indented, with stomatitis, which he overlooks or diagnoses and treats as ptyalism, and disregards the severe wasting diarrhœa with increased knee-jerk, pupillary anomalies, marked atheroma, enlarged epitrochlears, dorsal pain, and "nervous dyspepsia." I am compelled to say that cases of so-called postoperative insanity admitted to this asylum point to this conclusion, for in the spring and summer of 1908 we had, as we do every year, many such cases which showed all these symptoms of early pellagra and gave histories of former eruptions and diarrhœa. To cite an unusual occurrence, but striking coincidence, in one week, we admitted five cases of pellagra which had been operated on for gynæcological trouble within the preceding two months, and all of these at the time of admission had or within a month developed the typical objective pellagrous stigmata. Three of these five died and two went home recovered, according to Dr. Saunders, of our State Hospital for the Insane.

It has been known for three hundred years that pellagra can and does cause uterine hemorrhage, varying from a mild to a very severe degree. Old women, for example, not only in this country but abroad, have had hysterectomy done for pellagrous hemorrhage under the

false diagnosis of cancer. In this connection I cannot resist the question, How many neurotic and much-operated-on women, the proverbial gynæcological cases, who have suffered from many physicians, including a D. & C., R. V. O., suspensio-uteri, etc., movable organs fixed, and fixed organs removed, still not relieved, may be chronic pellagrins? I have personal knowledge of a number of such cases. My experience is as follows:

Case I.—Mrs. —— aged 40, married; several children; was sent to the hospital for diagnosis and operation, if necessary. There was marked anæmia, loss of flesh, and emaciation; hæmoglobin thirty per cent. There were definite pains and rigidity over the pylorus. Blood in the stomach contents and in the stools. No tumor could be felt. The gastric analysis showed positive interference with motility, blood, entire absence of hydrochloric acid.

We considered such a case as having symptoms sufficient to warrant an exploration. This was done. All that was found, however, was a red and inflamed stomach and intestinal tract. The case was closed. Several days later, when the patient was rolled on the porch and placed in the sunshine, the typical eruption was brought out on her hands. Drs. Babcock and Watson were called in consultation, with the result that the case was finally pronounced pellagra, which diagnosis was correct. The patient was sent home and died several weeks later, presenting a classical picture of pellagra.

Case II.—A young, vigorous child, taken suddenly ill with acute abdominal pain, with general abdominal tenderness and rigidity; most marked over McBurney's point. Temperature 102°, pulse 120. Two pellagra experts saw this case and advised operation. On opening the abdomen the appendix was found slightly inflamed, but entirely inadequate to explain the severity of the constitutional symptoms. Five days later, when the eruption occurred, the real cause of the trouble was revealed. This case recovered.

Case III.—Mr. ——, a middle-aged man, with a history, extending over a number of years, of some chronic abdominal trouble; he had been under the treatment of numerous doctors, and I saw him in consultation with a doctor of unusual ability and who was an expert on the question of pellagra. For some time previous to the consultation he had developed symptoms that to us seemed to indicate clearly either a high-up chronic appendix or an inflamed gall-bladder. As every other means had failed to give relief, operation was advised and accepted; although the appendix was removed and the gall-bladder drained, all of us felt that what was found did not explain his trouble. He was kept in the hospital for a long time under observation. While there, he developed a very mild pellagrous rash on the back of his hands, which settled the question of diagnosis. Proper dietetic and hygienic treatment, directed by this medical attendant, put him in better condition than he had been for years.

This patient, a most intelligent and educated man, then wrote for us, in his own way, a history of his trouble for several years, and it fitted exactly into the symptom-complex of pellagra.

Another point of importance is that in the future we must reckon with pellagra as a causative factor in intestinal hemorrhage of obscure origin. We also have to look at these cases not only from the standpoint of diagnosis, but it is important to remember that these cases are living on a narrow margin of safety, and that even when surgery is imperative for other conditions that may arise, such patients are extremely poor surgical risks, and the mental symptoms can be, and have been, greatly exaggerated by anæsthesia; operation occasionally being followed by acute insanity, from which they never relapse, but die.

In conclusion, it is most important to keep in mind that there are both acute and chronic cases; cases with and without eruption. That the onset frequently is most insidious and atypical; that the disease is a malady as protean as malarial fever, tuberculosis, or hysteria. This final point we wish to emphasize that, as our knowledge of pellagra increases and the pellagra zone widens, we should keep in mind, when dealing with cases similar to those given above, the pellagra syndrome.

SURGERY OF THE THYMUS GLAND, THE THORACIC LYMPHATICS, AND THE VAGUS NERVES *

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THE THYMUS GLAND

The thymus gland is of more importance to the fœtus and infant, of less to children, and of none at all to adults. Its function is not known. Experimental work has not added much to our knowledge of this gland; possibly it is useful only during the nursing period, or, perhaps, to a greater extent *in utero*.

We find the following analysis of M. Friedlebin's work on the thymus gland in the *Medical Times and Gazette*, of July 23, 1809:

Anatomy.—1. The thymus is a gland without any external duct; it is composed of an infinite number of lobules, each one consisting of closed follicles, united together by a very fine cellular tissue.

2. Its nerves are the avenues of its vessels, and proceed from the ganglion of the sympathetic nerve.

3. The thymus has no free cavities; what have been taken for cavities are interlobular intersections.

4. It contains a secretion composed of a clear transparent fluid, holding innumerable round nucleoli in suspension, mixed with some cells.

5. The nucleoli pass directly into the veins of the thymus.

6. The follicles of the thymus continually perish and are renewed; the bodies which have been called concentric are only follicles during their metamorphosis.

7. The thymus increases continually from its embryonic origin up to the age of puberty; but its increase is relatively less than that of the body. Between the ages of fifteen and twenty-five it remains stationary. It begins to diminish in volume at the end of this period, rapidly decreasing in adult life. After this period the thymus is rarely met with; and in the form of fatty tissue.

* Read before the Western Surgical Association, December 18–19, 1911.

Experimentally the thymus gland has been frequently considered anatomically, physiologically, and surgically, but aside from its anatomical and surgical aspects little is known, nothing definite having been discovered in regard to the physiologic action. The glands of various forms of animal and insect life and their influences upon the sexual organs, the blood, the brain, and the nervous and digestive systems, have been investigated by numerous scientists, without in any way increasing our knowledge of the functions of the thymus in man.

I have personally found the thymus gland present in the following animals:

Reptiles	Birds	Mammals
snakes	English sparrow	rats
tortoise	chickens	mice
turtles	robins	cats
	pigeons	dogs
	larks	rabbits
	cat-birds	squirrels
	owl	calf
	hawk	sheep
	crow	pig

MEDICAL TREATMENT OF AFFECTIONS OF THE THYMUS GLAND

Radium, being known to have a specific therapeutic effect upon neoplasms, has been suggested in the various pathologic conditions of the thymus. It should be applied directly to the gland at intervals of varying time and duration by means of thoracotomy or thorectomy by flap or trephine.

Serum treatment of thymus hypertrophy has not been carried beyond experimentation, but as it has been beneficial in the treatment of other glandular affections it is reasonable to suppose that it will be found useful, if not curative, in this class of disease.

SURGERY

We may have the thymus gland to reckon with surgically, even in adults, as its remnants sometimes undergo changes of a more or less serious pathological nature. It is, however, of greatest surgical importance in the first fifteen years of life. Operative intervention may be necessary because of injury or disease. The

effects of an enlarged thymus are almost purely mechanical. The sternum may be pushed perceptibly forward, or the neck upward, or both conditions may exist at the same time. The gland may extend backward and downward or laterally, depending upon its size and density and the resistance of the sternum and soft structures of the neck, and may exert pressure on the blood-vessels, trachea, or pneumogastric nerve. It is surrounded by a loose capsule, which may become distended by exudation.

Status lymphaticus does not always necessarily mean that the gland is enlarged; it may or may not be so; nor does enlarged gland necessarily indicate status lymphaticus.

Lang says, "Drawing my conclusions from cases recorded in the literature, thymus deaths may be grouped under three headings. The first group includes those cases in which there are actual pressure symptoms appearing some time before death and the postmortem reveals evidence of pressure upon the mediastinal contents. In the second group, the thymus, while enlarged, does not produce any prodromal pressure symptoms during life, and the postmortem shows enlargement, but no actual signs of pressure. The fatal issue in these cases is explained by a sudden swelling of the thymus due to some exertion, as in crying, or due to some interference with the circulation, as no doubt many of the undetermined origins of mediastinal pathology should be ascribed to the thymus gland or its rudimentary structures. Anatomical relations are changed so materially when large neoplasms are present that it is absolutely impossible to assign their origin, especially after the glandular structures have become absorbed as the result of age or disease. Spontaneous cure having not been infrequent, ante- and postmortem appearances are so greatly at variance that much time and observation will be necessary to determine certain facts. Color, consistency, size, area involved, and bloodless conditions are markedly different in death than in life."

Intubation is resorted to as the simplest palliative measure when breathing is interfered with, and may be performed with a rubber catheter if other means are not at hand. Intratracheal rubber tubes are also effectual. It is important to make sure that the tube opens at the end and not on the side. Introduction may be accomplished without either local or general anæsthesia.

Tracheotomy is second in choice if intubation is not practicable. It is frequently necessary to extend the tube well into the trachea, in some cases even to within an inch of the bifurcation of the bronchial tree. The operation should be done with local anæsthesia, but in an extremity the anæsthetic may be dispensed with.

Thymotomy is performed for cysts containing blood, pus, or serum, and for the removal of foreign bodies. A median incision is made, extending upward from the sternal notch, and the soft parts are divided by blunt dissection down upon the gland. The capsule is then opened and the gland lifted out. If no lesion is found, and there is no evidence for further direct surgical procedure, the gland may be returned, and the opening in the chest closed with or without drainage, usually the latter.

Thymopexy.—With the presence of a cyst of any character the lips of the incision in the gland may be fixed in the thoracic opening by suturing the gland and its immediate tissues to the edges of the soft structures of the wound, so that drainage may be continued indefinitely. This procedure will often relieve all symptoms.

Thymorrhaphy.—Suturing of the thymus is done for incisions, lacerations, ruptures, and punctures. Small, heavy, sharp, curved needles, carrying fine silk or linen or long-lived catgut, as small as can be conveniently supplied, are used, and as much tissue as possible is included in the sutures, which should be uninterrupted.

Thymectomy.—If extirpation is necessary, the blood-vessels are ligated before they enter the upper pole, and the gland is removed with little difficulty. Since Rehr (1896), who was the first to remove the thymus, the operation has been performed a number of times, improvement, as a rule, following. The mortality has been slight. Ransohoff states that he saw a patient from whom Durante (1906) had successfully removed a sarcoma of the thymus, including a portion of the innominate artery one inch in length.

PATHOLOGY

The thymus gland is as versatile as any other tissue of the body except the cutaneous structure. This is evidenced by the presence of congenital hypertrophy, abscess, cysts, tuberculosis, syphilis, angioma, nævus, hemorrhage and hæmophilia (direct or indirect), epithelioma, myxœdema, lipoma, fibroma, lymphoma, and various

other types of sarcoma. Any of these conditions may be found in infant or adult life; when in the adult a belated gland has been the cause.

THORACIC LYMPHATIC DUCTS AND GLANDS

Experimentally, the general study of the lymphatic system of the thorax has been extensive and satisfactory. That portion pertaining to progressive glandular infection, however, remains quite undeveloped, as, for instance, the reason for the lack of uniformity in the glandular involvement by malignant growths. If the accepted rule of extension predominated, all malignant growths upon or within the thorax would become general so that there would be no necessity for resorting to surgical measures of any kind or degree.

Bartiolinus (1653) was one of the earliest to experiment upon the thoracic duct, while Cooper (1798) mentions three instances of obstructions to the thoracic duct with experiments, showing the effects of tying that vessel, and Amussat (1840) mentions an extraordinary development of the thoracic duct. Uspensky (1888) records most interesting experiments by ligating the thoracic duct to determine the effect on chemical and morphological substances of the blood. Sryeff (1889) ligated the duct of a dog to determine the effect upon nitrogenous metamorphosis, and Toushinnikeff (1891) ligated the duct to determine its effect on exchange of gases in animals.

SURGERY

Not only glands, but terminals, must be considered in the surgery of the lymphatics, because infection of the glands and channels occurs alike.

The history of surgery of the thoracic lymphatic system is of recent origin, one of the earliest reports being that of Keen (1894), who gives a résumé of four cases of wounds of the thoracic duct in the neck, all having been operated upon. Jesus (1895) records a case of ligation of the thoracic canal. Schminn (1896) mentions an injury to the thoracic duct at the root of the neck during a surgical operation. Nattan–Larrier (1897) describes the technique for ablating the thoracic duct, while Cushing (1898) reports a case of operative wound of the duct which he repaired by suture, and

Dianesly (1903) one in which he implanted the divided thoracic duct into the internal jugular vein with recovery.

Thus, there are in evidence sufficient data to warrant the assertion that the thoracic lymphatic ducts are subject to the same surgical principles that govern veins and arteries, and that it will not be long until the number of operations for their repair will be legion. They may be exposed for surgical repair, but it is necessary to approach them through the neck or by means of a trans-thoracoplasty. The rules for suturing blood-vessels obtain in surgery of the thoracic duct.

Hypertrophy.—The ducts and glands frequently undergo hypertrophic and degenerative changes, aggravated by irritation or infection, especially of tuberculous origin. All diseases of the respiratory system are more or less the exciting cause. Symmers (*New York Medical Journal*, May 20, 1911) says that in lymphatic leukæmia enlargement of the cervical nodes commonly occurs, in which event the blood examination usually suffices to establish the nature of the enlargement. But in pseudoleukæmia there are no characteristic alterations in the blood and microscopical examination of the affected nodes reveals no criterion by which the changes may be distinguished from lymphatic leukæmia or from those instances of diffuse hyperplasia of lymphoid cells in which tuberculosis as the etiological factor is presumptive.

Cysts and Ascites.—Cysts of the thoracic duct may originate within the wall, or the wall may become dilated to form a sac with a small opening connecting it with the main duct. They may be anomalous or congenital, or they may be due to injury or disease, involving any portion of the duct, and varying from the size of a small pea to several inches in diameter. They may rupture and the contents escape into any portion of the mediastinal, pleural, or pericardial cavity, perhaps even finding the way through the diaphragm into the abdominal cavity, such a case having come under my own observation.

Glands also may become cystic and may either remain so or rupture into the surrounding cavities. The contents of these cysts may become infected.

Chyle has been found occasionally within the mediastinal space associated with injury, disease, or anomalous conditions of the

lymph-channels, and when present it may become absorbed, if undisturbed, and the opening in the duct closed, or it may be removed by artificial means, such as aspiration by trephine of the sternum or through the intercostal spaces, or by open drainage through an incision in the intercostal space, trephine, or a cartilaginous flap. If the open method is adopted, smaller lymph-channels which are found to be injured may be ligated; when the ductus thoracicus is ruptured it may be repaired by suture or closed by ligature. The thoracic wall is then closed and drainage maintained indefinitely by means of tape or rubber tubing.

Cancer.—The importance of the lypmhatics in connection with malignant growths of the thorax and its viscera requires them to be considered surgically. Without the removal of glands associated with cancerous growths a cure is supposed to be out of the question. In operating for carcinoma we have always to deal with the following considerations: Is it possible for a malignant growth to exist without lymphatic involvement? If not, to what extent are the glands and lymph-channels involved? Are they involved as groups? Can one gland in a group be involved without all of that group being involved? Can one group be affected without all groups being affected?

Carcinoma and lymphosarcoma are two of the most common forms of cancer involving the thoracic duct and lymph-nodes. They occur both primarily, and, more frequently, secondarily; primary involvement is often difficult to determine.

THE VAGUS NERVES.

Though the vagi have been described for several centuries, their function remained undetermined until a comparatively recent date.

Loget (1849) mentioned the nature and veritable usages of the various anastomosis of the vagi; while Jacquart (1864) described their pulmonary distribution in ophidilus. Langenbaker (1877) described the functions of the vagi in domesticated animals, and Stowell (1881) gave the results of experiments on the domestic cat.

Symptoms coincident with disease and injury of the vagi are not altogether defined. Many, no doubt, have been improperly ascribed, especially by the earlier investigators, whose opportunities were limited. Compression paralysis, neuralgia, neuritis, hypertrophy,

atrophy, trauma, and neurosis, together with the various methods of producing them experimentally, have been the means of forming reasonable symptomatic conclusions.

Schwarzenberg (1768) mentioned a rare form of spasmodic convulsions due to certain conditions of the vagi. Symptoms of pressure of aneurismal or other tumors upon the vagus and recurrent nerves are manifested by laryngeal disturbances, as suggested by Johnson (1875). Helm (1882) reported on anatomy and pathology of the nucleus of the vagi with reference to the theory of respiratory and cough reflexes of development and regeneration; and Habershore (1885) on the influence of cold shock upon the vagi.

Neuralgia.—Neuralgia of the vagi was described as early as 1828 by Pinel, since which time but little has been said in favor of its existence, because of the uncertainty of ways and means for its determination. The sensory fibres which enter into its construction are no doubt responsible for the more or less undetermined pain so often manifested within the thorax.

Pinel (1828) reported a case of neuralgia of the pneumogastric nerve, and Prus (1829), one in which he observed symptoms simulating hysteria. Schupmann (1840) reported a case of neuralgia of the vagus, and Lederer (1860) reported one similar in character.

Pressure.—Pressure of neoplasms and anatomical irregularities, both congenital and acquired, upon the vagi have been known to produce physiological disturbances of the organs which they control. In the *North American Archives of Medicine and Surgery,* Sc. Balt., 11, 172, 175, 1835, are mentioned cases of asthma, aneurism of the arch of the aorta, disease of the vagi and phrenic nerves, and large glandular tumors compressing them, hepatization of the lower part of both lungs and death from smallpox.

Herard (1846) reported a case of perforation of the bronchus and compression of the vagi due to tumor. Worthington (1855) records a death due to pressure of a tumor upon the vagus nerves, and Schwing (1857) one due to pressure from a stenosed foramen. Sir William Gull (1859) reported a case of death due to destructive changes of the lung from diseases in the mediastinum invading or compressing the pneumogastric nerves and pulmonary plexus. Potain (1861) considered at length hypertrophy of the bronchial ganglion and its influence upon the vagi, and Hittner (1865) stated

that a tumor involving the vagus caused peripheral and reflex pain. Stackler (1882) contributed an essay in which he claimed that epileptic convulsions were due to compression of the vagi, while Chapin (1884) reports a case of death from pressure on a pneumogastric nerve. Grande (1907) reported a case of compression of the vagi, and Oppert (1907) one of death due to pressure of a tubercular cyst.

Neuritis.—Neuritis of the vagi is probably of frequent occurrence. It may be primary or secondary, acute, subacute, or chronic, localized or general, and due to disease and injury. Rosch (1857) and Weiss (1842) each wrote upon the subject of inflammation of the vagi. Browne (1896) and Madir (1898) reported cases which they believed to be of this character. Perna (1904) refers to inflammation of the inferior laryngeal nerve. Neuritis affecting the perineum or sheath results more frequently with an infected wound than without it, but the nerve-fibres may even then escape serious damage. If the infection extends to the endoneurium, the nerve-fibres will become more or less affected. Pathological exudate is always to be considered more or less seriously, especially in the parenchymatous form, for the disease originates in the nerve-fibres.

Hypertrophy.—Cook (1857) reported his observations on the ganglionic enlargement of the vagus—the probable function of the ganglion, the position it occupies in the human subject and in several of the lower animals. Hypertrophy of the vagi or bronchial ganglia has been observed in both animals and man, and is probably due to injury and disease. It may be localized or general.

Neurosis.—Neurosis of the vagi was considered an independent disease by Blufus (1842), Sherman (1856), and Lederer (1866). Rupier ascribed its origin to the brain, and Forres (1877) wrote on functional disturbances due to neurosis of the vagi. Such conditions have been verified by more recent observers.

Traumatic Pressure.—Pressure from depressed fracture of ribs and cartilages or vertebræ has been proven to be effectual in causing functional disturbances, or from foreign bodies of the vagi. Herard (1851) reports a remarkable case of asphyxia produced by a traumatic compression upon the vagi and sympatheticus.

Pain.—Pain in the vagi is reported by Anderson (1874), who

states that it was of a burning character in the fauces; that it was attributed to the vagus, and that it was relieved by atropia.

Cancer.—Secondary cancer of the vagus due to infiltration of the neck is reported by McCollum (1869). Primary neoplasms of any character are indeed rarely, if ever, found in the nerve. If so, they have not reached the ordinary literary channels, though Taylor (1885) reported having observed a calcareous deposit in the vagus.

Paralysis.—Paralysis does not always result from severing or even excising portions of one or both vagi, though it quite commonly occurs, probably depending upon the point of severance. The sympathetic dominates after severance.

Paralysis of the vagi was reported by Coffier (1882) and Holzknecht and Hoffbraur (1907); also Stenhaur (1908).

SURGERY.

Moreau (1877), by experiments upon white rats, found that regeneration with complete restoration of function could be obtained after the vagi had been divided. His experiments were confirmed by Philapeaux during the same year. New nerve element forms centripetally from the proximal end, thus indicating that the ends of divided nerves must be united. When not completely severed, the time required for regeneration is six to eighteen months, depending upon the distance of the injury from the peripheral endings of the nerve.

Protopathic return earlier than epicritic sensibilities, the latter requiring two years. The area of the last sensibilities is greater in the protopathic form. Divided nerves do not lose their function to conduct impulses; therefore do not undergo complete degeneration.

The pneumogastric nerves may be lacerated or severed by injury or surgical operation, and thus require anastomosis by suture. The severance of one need not require immediate anastomosis, while the severance of both would probably result in too rapid death to permit of successful anastomosis. If one or both are divided by surgical operation while the chest is open they may be immediately united and the life of the patient perhaps saved. Double vagotomy has been done in dogs without causing death.

Experiments upon the vagi are of ancient origin. They have

been made principally to determine their physiological effects upon the organs which they supply, but experiments to determine their surgical possibilities are of more recent origin. Stretching, together with the internal administration of various drugs, has been a selected, experimental method to determine certain functional results.

Compression.—Persistent pressure will partially or completely destroy nerve function. It may be applied by means of gauze, ligature, instruments, or may be caused by foreign bodies, neoplasms, or fractured vertebræ, ribs, or cartilages.

Compression of the vagus nerves to determine its effect upon the respiratory and digestive tract was done by Depuy (1816 and 1821) and Mayer (1827). Habershon (1864) illustrated the effects upon aneurismal tumors after implication of the bronchus and the vagus nerves.

Navroski (1870) studied the effects of blood-pressure on the vagus nerves, and Waller (1870) compressed the nerves to produce asthenia or anæsthesia in surgical operations. He also compressed the vagus nerve for the cure or relief of various nervous affections.

P. Dix (1872), after removing a tumor from the inferior carotid triangle, noticed considerable functional disturbance of the vagi, and Johnson (1875) recognized laryngeal symptoms resulting from the pressure of a tumor upon the vagus and recurrent nerves. Salamon (1880) reported suppurating bronchial pneumonia resulting from pressure of the vagus due to aneurism of the brachio-cephalic. Francois-Franck (1886) discussed at length the communication of Lafont upon the various effects of compressing the vagus.

Boix (1893) produced tachycardia by compressing the vagus. Dubois noted the therapeutic effects of compression of the vagus nerve, and Marklin (1893) compressed the vagus to produce systolic changes of the heart. Walraveus (1896) compressed the vagus nerves to determine the effects upon urinary secretions.

Vagotomy.—Vagotomy may be done by incision, ligature, cautery, or laceration, such methods having been employed in both animals and man, at every point throughout its course. Ware (1828) performed vagotomy to determine its effects upon the heart and lungs. Bonley (1852) made a section of the vagus to determine the effects upon absorption of the stomach, and Boddart (1862) the effects upon the lungs after section of the vagus nerves.

Roddaert (1862) reported his experiments upon consecutive section of the vagi for pulmonary lesions.

Krishaber (1869) reported his observations on the phenomena provoked by chloroform after section of the vagi, and Boddart (1877) some remarkable observations upon the results of simultaneous section of the vagi in the cervical region.

Francois-Franck (1879), after injuring a vagus nerve with a ligature and contusion, restored its function by electric neurotomy.

Vaulair (1893) made successive division of the vagus without causing death, but Pavloff (1894) produced death of animals by section of the vagi. Martini (1894) made vagotomy and then sutured the ends together. Pavloff (1895) severed both vagus nerves of dogs to determine the results other than death. Herzen (1897) performed double vagotomy without death. Cheskoff (1900) vagotomized dogs with respect to circulation, respiration, digestion, and heart regulation, and Ribera (1901) sectioned the vagus for a similar purpose. Marcenghi (1901) reported his observations of the effects of bilateral vagotomy upon respiration.

Nikolaides (1901) demonstrated the general effects of vagotomy, while Crile (1902) studied the effects of severing and mechanically irritating the vagi. Gomes (1903) made a double vagotomy, and Fronis and Paferski (1904) made an intrathoracic section of the vagus in the abdominal cavity. Martini (1904) performed vagotomy upon dogs, and Luszkai (1905) anastomosed both vagus nerves and sympatheticus after they had been severed for experimental purposes.

Starch (1904) did an intrathoracic vagotomy, while Firori (1904) considered the surgical possibilities of the vagi. Stewart (1905) made simultaneous section of both vagi above the origin of the recurrent laryngeal nerves, and Cannon (1906) studied the motor activities of the alimentary canal after splanchnic and vagus section. Cannis (1909) observed the alterations of myocardia after vagotomy, and the same year reported a case of the survival and regeneration of the vagus nerves after double vagotomy, and Jacquet reported a case of accidental section of the vagus.

Vagectomy.—Segments of the pneumogastric nerve were first removed for experimental purposes by Cruveilhier (1831) and Henkel (1833). Since then many similar operations have been made upon both animals and man for various purposes.

Claude Bernard (1849) produced paralysis of the œsophagus by resecting the vagus without producing death, as did also Bieroliet (1857), while Kappeler (1864) extirpated about one-half inch of the vagus for laryngospasm. Wood (1874) produced suspension of intestinal secretion and failure to produce purgation after section of the parvagum; and Vogel (1883) reported on the prognosis after vagus resection; while Arthud and Butte (1889) reported the results of their researches upon the effects produced upon the diaphragm by resection of the vagus.

Ramverida (1891) extirpated a vagus containing a sarcoma. Widner (1893) resected the vagus nerve, and Giordana (1893) considered the lesions of the vagi that might become surgical, and Park (1895) determined the results of resection of the vagus and phrenic nerves.

Preisz (1898) removed the vagus recurrens nerves in a case of chorea laryngis, and Marenghi (1898) recorded his observations on neurotomy of the vagi with reference to the problems of regeneration of their peripheral fibres.

Kachkavski (1899) excised simultaneously the vagus nerves in the necks of dogs without destroying their lives. Mollard and Regard (1899) reported on the lesions of the heart muscles and Cervera (1901) resected the vagus to determine the results upon the function of the heart and trigeminal nerves, and the result of consecutive resection of the vagi. Ladoue (1900) communicated the phenomena of chromatolysis resulting from resection of the vagi. Cheshkoff (1902) observed the lives of dogs for a year and seven months after simultaneous excision of both vagus nerves. Ruddel (1902) resected the vagus nerve, and Veavt (1907) reported resection of the pneumogastric nerve for a primary sarcoma.

Tuffier (1907) considered the proposition of resecting the vagi, while Broechkaert (1907) wrote upon its technique, and Murcell (1908) resected the right vagus nerve containing a malignant neoplasm. Decknoff (1909) resected the vagus nerve in man, and Dechonoro (1909) also resected the vagus nerve.

Stretching.—Stretching the vagus nerve in animals has been done for many years for physiological purposes, but it was not done in man until 1898, when Jaboulay did it to relieve certain intrathoracic pains.

Vagorrhaphy.—Suturing may be necessary to unite the severed ends of the nerve. It can best be done with silk or catgut, when the chest is opened under intratracheal insufflation or in the vacuum chamber. It may be immediately necessary when the nerves are severed during a surgical operation upon the thoracic viscera, or when they are severed by injury.

Vagorrhaphy is said to be primary when the suture is applied immediately or soon after partial or complete severance, and secondary when employed for anastomotic purposes after considerable time has lapsed.

Phillipaux and Vulpian (1863) were the first to demonstrate by animal experimentation that nerves can be united by suture without loss of function. This means of union is best accomplished with fine needle and silk suture, because of its superior strength and small size. For the absorbable materials, such as catgut and kangaroo tendon, to have the same strength and durability would necessitate an increase in the size of both the ligature and the needle to carry it through the nerve with the resulting increased trauma. With normal nerve-tissue a needle without cutting edges can be easily inserted, but with the presence of cicatricial nerve-tissue a cutting edge is necessary, especially when there is dense neuromata. With secondary suture function is usually incomplete because of atrophy. The nerve may have become permanently shortened, thereby causing great permanent loss of function.

If electrical stimuli are effective, motive power may be benefited by constant current. Sherren believes that the length of time of separation of the ends is of less importance than infection of the wound. Wertheimer (1901) anastomosed a vagus nerve in the thorax of the human being.

Miscellaneous Surgery.—In this class are found much interesting data pertaining to the general surgical possibilities of the vagi.

Ware (1828) imposed trauma upon the vagus nerves to determine its effect upon the lungs and stomach, and Laffout (1836) injected chloral hydrate and chloroform into dogs intravenously to determine the effects upon the heart and vagus nerves.

Fearn (1847) reported a case of gunshot wound of the internal carotid artery and divisions of the vagus. He ligated the artery, but did nothing to unite the ends of the nerve. Recovery.

Blake (1865) reported a wound of the neck, apparent recovery; sudden death. Boulland (1891) records a case of mortal injury to the vagus nerve. Djelicoff (1901) contributed to the study of accidents to the heart of the vagi.

Giordano (1893) experimented on surgical lesions of the vagi, and Langley (1897) made a series of experiments about the junction of the vagus nerve with the cells of the superior cervical ganglion, and Cesipoff (1898) in the region of the central origin of the tenth pair of crural nerves.

Powers (1903) removed a heavy silk ligature that had been applied to the right common carotid artery, and which unfortunately had included its corresponding vagus nerve for more than one year. He also reported clinical symptoms before and after removal.

The after-care for sutured vagi should, as a rule, when operated on by way of the neck, be drainage for twenty-four hours when not primarily infected, and continuously when infected primarily and secondarily. Quietness, liquid diet, and recumbency should always be insisted upon. Soiled dressings should be changed frequently, but irrigation should be seldom employed.

Surgical Technique.—Transcervical Method.—Vagotomy, vagectomy, stretching, vagorrhaphy, and the direct application of the needle for electrolysis or the injection of medicaments are done in an incision through the skin and sterno-mastoid muscle at the level of the cricoid cartilage. The deep jugular is turned aside and the nerve separated from the common carotid artery and sheath. Great care should be exercised to prevent injury to the vein and artery.

Transthoracic Method.—After severing the ribs at their sternal junction or removing the sternum, the vagi may be seen in the superior mediastinum behind the right innominate vein on the right side of the trachea.

Trans-abdomino-diaphragmatico-thoracic Method.—The upper abdominal wall having been opened, the diaphragm may be incised one inch to the left of the gastro-œsophageal junction, when the vagi may be observed with but little difficulty.

ANORECTAL FISTULA *

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GENTLEMEN: The first patient, a colored man, aged forty-eight, was admitted to the hospital a few days ago suffering from anorectal fistula. He comes before us to-day for operation, which it is hoped will give him entire relief.

When the patient entered the hospital he complained of severe pain in the anorectal region, and inspection revealed a sinus about an inch to the left of the anal verge, from which emerged pus in considerable quantity. He told us there had occurred what he thought was a "pile" in this region some time before, that it was painful for several weeks, and finally ruptured spontaneously, the pus making its exit, according to his statement, further out toward the buttock. No history can be obtained to indicate that there is an internal opening to this sinus; but it must be remembered that the history given by patients of this class is of little value, and in most instances a complicated condition of affairs is found when an attempt is made to excise the fistulous tract. From the information thus far obtained, it is believed there exists in this case a blind or incomplete fistula, *i.e.*, one having only an external opening.

The preparation of fistula patients for operation is a matter of the greatest importance, particularly if the wound is to be closed by immediate suture, as can sometimes be accomplished where there is a simple fistula with only an external opening. In these cases no fecal matter must be permitted to come in contact with the wound surface, and in order that this may not occur the object is to have the bowel completely empty. To accomplish this, the patient is given a purgative the second night preceding the day of operation, and a large soapsuds enema the following afternoon. After the purgative has

* Clinical lecture delivered before the senior class of the University of Louisville, Medical Department, at the Louisville City Hospital.

acted thoroughly, the enema clears the bowel of any remaining fecal matter, together with any mucus or pus which might contain pyogenic organisms. Another soapsuds enema is given three hours before beginning the operation, in order that no retained water, which might contain fecal matter, colon bacilli, or other organisms, may pass over the wound surface. Any water which may have remained within the folds of the rectum will have been absorbed during the three hours. It is important that the foregoing rules be followed as nearly as possible; but sometimes in hospital work, where necessarily the assistants are very much hurried because of the large amount of work to be done, the procedure suggested cannot always be implicitly followed, and instead of a perfectly clean operative field there may be fecal matter to contend with.

The patient now being anæsthetized, a grooved director is inserted into the sinus, and with a finger in the rectum pressure is made toward the probe to determine the extent of the disease: whether the sinus extends into the rectum, or whether there is merely a blind fistula which loses itself in the tissues. With the finger in the rectum exerting pressure upward, pus emerges from a second opening near the median line. This is an unexpected complication and will probably make inadvisable any attempt to close the wound by immediate suture. At this point in the examination attention is directed to an important feature, viz., with a finger in the rectum and the thumb outside, the indurated tissues being thus brought between them, one can judge as to the extent of the disease with considerable precision, and in this way approximately determine the amount of tissue which must be excised.

With knife or scissors an incision is first made through the indurated tissues upon the grooved director, and the sinus will then be completely excised or thoroughly curetted, as may seem best. No internal opening has yet been demonstrated. Attention is called to the character of the tissue through which the incision is being made; it is markedly indurated, lifeless, and almost gangrenous. The tissues tear easily and can be separated with the fingers, they are so friable. Several branches of the main sinus are found extending in various directions, rendering immediate suture of the wound entirely out of the question. All the overlying tissue and skin will therefore be carefully excised, and the wound permitted to heal by granulation.

Where a wound is to be left open, as is necessary in cases of this kind, the edges must be carefully trimmed away, thus making as nearly as possible a flat surface, in order that the skin may not come together before the bottom of the wound has been filled by granulations. It may appear to some of you that an unnecessary amount of tissue is being sacrificed in completing this operation, but it must be remembered the operation must be radical to be permanently effective. If any diseased tissue or a small fistulous tract be left, the operation might as well not be performed, as the patient would derive no benefit therefrom.

As has been repeatedly emphasized, in operating for anorectal fistula, where extensive excision of tissue becomes necessary, one must bear in mind that there are about the rectum certain tissues which may be incised and removed with impunity, and certain others which may not. The incision may be extended far out on the buttock if necessary, and a considerable portion of the adipose tissue in the ischiorectal space may be safely removed, but if the sphincter muscle be badly damaged the patient will almost necessarily be left with partial or absolute incontinence.

Quite a large separate and distinct sinus found in the left posterior quadrant is now being divided and excised, which shows that at least two abscesses were present in this case. There is no reason why multiple abscesses in the anorectal region may not develop simultaneously, particularly if the patient is a tuberculous subject, and, indeed, a large percentage of ischiorectal abscesses are tuberculous in character, which is believed to be true in this case. Oftentimes it may be thought there exists a simple uncomplicated external fistula, and when incision is made a complicated condition of affairs is revealed. What appeared, from external observation, to be a simple sinus is found to lead into another, perhaps more extensive, channel; sometimes the entire rectum is surrounded by fistulous tracts extending upward just underneath the mucous membrane outside the rectal wall, or far out into the adipose tissue of the ischiorectal space. One may think he is dealing with a simple fistula and proceed to excise that part, then another sinus is discovered and excised, until finally a large cavity has been made; but if the directions previously mentioned be followed, *i.e.,* to remove only such tissues as can be spared, being careful not to damage those which must not be disturbed, even though

a secondary operation may in some cases be necessary to secure a perfect result, the operation may be undertaken with the greatest degree of confidence and safety. On the other hand, sometimes alarming symptoms may be present and the appearance of the patient indicate a serious condition, yet incision may demonstrate that the fistula is very superficial, that the pus formation is diffused just underneath the skin, and that the incision need be extended no deeper than the fascia or muscular structure.

With one finger in the rectum pressing outward toward the sinus, using scissors curved on the flat, the diseased tissue can be rapidly excised. After having performed a few unsatisfactory operations of this kind, every surgeon better understands and appreciates the importance of making a large wound. Remember, the tissues rapidly contract, and what may seem a large surface at the time of the operation will in a few days have contracted to a marked extent.

While we have already made rather free use of the scissors in excising the tissues involved in the numerous sinuses discovered, attention is called to the dark color of some portions of the wound surface, which indicates the necessity of going still deeper to reach healthy tissue. This may be accomplished with a curette if preferred, but the better and quicker method is to use the curved scissors.

Careful search has been instituted and no additional sinuses or branches can be located, and all the diseased tissue has been carefully eliminated. There now remains the typical wound resulting from the excision of an anorectal fistula. All the hemorrhage during the operation has been controlled except the slight capillary oozing.

While the dressing is being applied, a few words may be appropriate concerning the after-treatment. The dressing consists of sterile gauze. Iodoform gauze is still used by some operators, but it is believed inadvisable in cases of this kind. Only after operations for extensive tuberculous fistula should iodoform gauze be utilized, and even then its reputed advantages are seriously questioned. Sterile gauze "fluffed up," *i.e.*, not inserted flat, should be tightly packed into each cavity, then covered with a pad of flat gauze, over which is placed the perineal bandage. A band is first placed around the abdomen just above the hips so it cannot slip downward, then brought forward to one side of the scrotum over the anal gauze pad, then upward under the abdominal band and back over the anal pad on the

opposite side of the scrotum and securely fastened in front to the abdominal bandage. In the event the material for a bandage of this kind be not available, an ordinary "T" bandage may be used, but it is impossible by this means to secure as firm and even pressure over the anal region as with the perineal bandage described.

Before the patient is put to bed, he is given a hypodermatic injection of one-fourth grain morphine with 1/150 grain atropine. This is for a twofold purpose: (*a*) to control pain, which ought not to be great in a case of this kind, and (*b*) to prevent too early movement of the bowels. Another hypodermic of one-quarter grain morphine may be administered later if pain is sufficient to warrant it. In twenty-four hours the outer bandage is removed. The gauze packed in the wound is left intact for about forty-eight hours. It is then saturated with a fifty per cent. peroxide of hydrogen solution, to soften and assist in detaching it from the raw surfaces, when it will come away without any bleeding of the wound. If an attempt is made to remove the gauze packing by simply saturating it with plain water, pulling at it, etc., the capillary vessels will most likely be opened and hemorrhage result, which will make the gauze more difficult to remove. The wound is again packed with gauze, less tightly than before, and another anal pad applied.

After four or five days, when granulation has commenced, at each dressing the gauze is saturated with a mixture of balsam of Peru and castor oil. This prevents sticking, keeps the wound soft, hastens granulation, and stimulates healing. Sometimes exuberant granulation tissue develops in the wound, and when this occurs its removal becomes necessary. Sulphate of copper is ordinarily employed for this purpose, but powdered burnt alum is also excellent. It is necessary that the wound heal from the bottom, the sides not being allowed to come together, otherwise there might be left a cavity underneath which would require a secondary operation.

Although in the case before us considerable tissue has been removed, when granulation has taken place and the wound has completely healed there will be only a slight deformity. In the process of granulation the wound fills out, resulting in normal conformation of the surface. It is anticipated, unless some unforeseen accident occurs, that this patient will be entirely well and ready to leave the hospital in less than three weeks.

The next patient, male, aged forty-two years, was operated upon two weeks ago for a complicated anorectal fistula, and is exhibited for the purpose of showing the after-effects of the operation. While there remains considerable soreness about the rectum, the wound is granulating nicely, and the patient complains of no pain. It will be noted that the dressing in this case has been fastened in position with strips of adhesive plaster, which is an excellent method for use after granulation has progressed for a week or two.

There is a slight discharge near the middle of the wound, which indicates that a small portion of the sinus was evidently not completely excised. This emphasizes what has been previously said, *i.e.*, the necessity of trimming off all redundant tissue and thoroughly excising every fistulous tract in its entirety, no matter how insignificant it may appear at the time. While the edges of this wound were carefully trimmed at the time of the operation, it would have been better had more tissue been removed. However, the indications point to a successful result. The edges of the wound are still somewhat swollen and tender.

There is another point worthy of mention in reference to this case: According to the history obtained, while in a toilet room about four years ago, this man suffered a very considerable hemorrhage from the rectum. From what source the blood actually came it is now quite impossible to determine. It was probably due to one of three causes: (1) internal hemorrhoids, (2) rectal polyp, or (3) a so-called capillary pile. Where hemorrhoids exist entirely within the rectum, if the patient become constipated passage of the hard masses of fecal matter may produce rupture of a small blood-vessel, resulting in extensive bleeding. In the pedicle of a rectal polyp there are always two blood-vessels, an artery and a vein. Such a polyp may exist in the rectum for a long time without the patient's knowledge. Until the pedicle becomes sufficiently elongated to enable the polyp to be grasped by the sphincter muscle, its presence may not be suspected. The polyp is constantly pressed downward by the fecal matter, especially when the patient is constipated, and by gradual elongation the pedicle becomes weakened, finally tearing off, and hemorrhage follows. There may be present a so-called capillary pile (which is really not a hemorrhoid but a nævus) without the patient's knowledge, until examination of the rectum is made because

of the occurrence of sudden hemorrhage. I have observed several examples of this kind. In one the primary hemorrhage, which occurred without warning, was so great that the individual when seen was pulseless and apparently almost bloodless. A so-called capillary pile is simply a collection or tuft of blood-vessels (arterioles and capillaries) upon the surface of the mucous membrane of the rectum. The passage of hard fecal matter, or a foreign body such as a piece of bone partially embedded in the fecal matter, may abrade this tuft, thus opening the blood-vessels, and, on account of the limited contractility of the rectal mucous membrane, hemorrhage continues until the blood-pressure of the individual becomes so reduced that it finally ceases simply for want of pressure from behind.

The last patient, male, aged thirty-eight, was operated upon for anorectal fistula one week ago to-day. The beginning of tissue granulation is readily observed. The wound is not as red as in the case of the man just exhibited, who was operated upon two weeks ago, yet there is sufficient color to indicate a healthy granulating surface. The wound is healing nicely from the bottom, the discharge present is normal in amount, contraction is gradually taking place, and every indication points to an entirely successful ultimate result.

Gynæcology

SOME PLASTIC VAGINAL OPERATIONS

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Any undue or prolonged stretching of the intrapelvic soft structures, during parturition in its second stage, is as bad in its subsequent results, and as serious in its effects, as a pronounced laceration of the parts; so also it may be as difficult to well repair. It is not out of place, I think, to speak in short of the frequency of the resulting symptoms; nor is this to be wondered at, when we recall to mind the not uncommon bad relationship of the fetal head, in its diameters and in its varying obstetrical positions, to the pelvic canal. I will not review the subject in all of its details of forms and degrees, but will merely give attention to the subject of cystocele—its frequency, its mechanical causes, its results, and what appears to me to be a judicious method for its correction.

A ruptured or stretched perineum and posterior vaginal wall are by no means of infrequent occurrence. If the condition is not corrected at the proper time, or normal relations are but imperfectly restored, we have a gaping vulvar orifice, delayed involution of the parts, descent of the posterior vaginal wall, in time sagging of the anterior vaginal wall, and finally the formation of a cystocele and a rectocele.

Almost all cystoceles are directly dependent upon perineal lacerations and stretchings. The anterior vaginal wall lies close to the posterior bladder wall and moves with and supports it. If this support is weakened, the bladder, disordered in place in its posterior wall, cannot normally and completely empty itself, and sooner or later becomes irritable, from the effects of residual urine, *i.e.*, ammoniacal fermentation and decomposition. In point of frequency there

is but little question that this condition stands next to a gonorrhœal infection, as a causative factor of frequent, painful, and unsatisfactory micturition in women. If the urine be examined it will be found that, while there is no special abnormality of specific gravity, it is at times alkaline in its chemical reaction. While it is not albuminous, yet when it is held, in a vial containing several ounces, to a strong light, there will be detected more or less flakes of bladder casts, indicative of an exfoliation of the mucous membrane.

In the treatment of this condition internal medication has little value. An abundance of wholesome water, particularly Vichy or lithia water, does good, but will not cure, because underlying causes are not corrected. Permanent relief can only be obtained from a steady improvement of the mechanical abnormality of the bladder.

In the performance of an anterior colporrhaphy, a procedure usually necessary to effect a cure of this condition, I would suggest the following. From it I have secured very good results.

After cleansing the vulva and the vaginal canal with green soap and with lysol water (℥j to Oij), introduce within the vagina a large Simon's univalve speculum, to retract the perineum and the posterior vaginal wall, so as to expose to full view the anterior vaginal wall with its cystocele formation. The patient should assume an exaggerated lithotomy posture. It is not necessary always to draw down the uterus to full view; if it is high within the pelvis and atrophied from age do not disturb it.

1. Seize the cystocele with a hæmostatic forceps two-thirds of the way up the vagina and in its centre, and draw the mucous membrane downward and well over to the left. Then with knife, or better a pair of scissors curved slightly on the flat of the blades (the tips turned away from the interior of the bladder), detach the mucous membrane of the vagina, deep and high, in a semicircular line, from the right toward the left. So much for the first half of the area of the dissection required.

2. Seize the remaining portion of the cystocele in the median line and centre with another hæmostatic. Draw its mucous membrane well over to the right; then dissect this fold entirely away from left to right. There will then be made a full area of denuded tissue, symmetrically oval in shape. Hæmostatics right and left, as well as above and below, are of great assistance and define accurately the

proper outlines. Make the area of denudation long, wide, and deep, into the cystovaginal muscularis; or, better, through the same, to the bladder mucosa itself. The denuded tract should be broader above than below, and should extend to the cervix uteri. If the cystocele is large and of long standing, it is well indeed to extend this denuded area above onto the anterior face of the cervix, and laterally onto either side of the same. We avoid injuring the bladder by turning the tips of the scissors away from this organ.

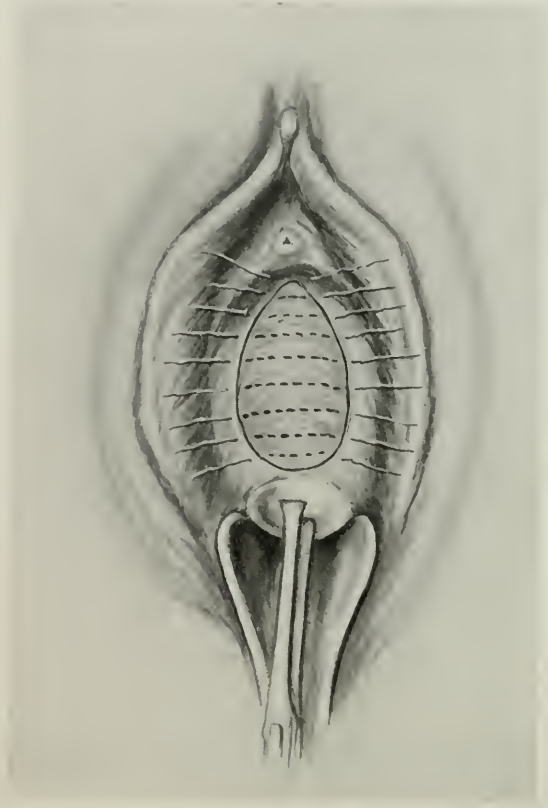
Sutures of good catgut may be arranged in the continuous or the transverse form. If the latter is chosen, from six to twelve may be required to coapt the incised edges thoroughly and firmly (Fig. 1). After the sutures are all in position, each one grasping an abundance of the lateral adjoining edges of undenuded tissue, it is well to push upward into the bladder, as far as possible, the protruding mucous membrane of the cystocele, before they are tied. The life of catgut, for all operations, ought to be at least two weeks. Rarely will any vessels need tying in this operation.

When a perineorrhaphy and posterior colporrhaphy are done at the same time (generally necessary), they should be performed after the Hegar or the Emmet method, and in such a manner as to build up a new pelvic floor, thicker, firmer, and stronger than the natural one, in order to give sufficient additional support to the anterior vaginal wall. On this account a narrowed vagina is to be constructed, forbidding, it may be, sexual intercourse, but as the operation is usually done for women after the menopause, this deprivation will not be regretted when the correction of a dislocated bladder and relief from its attending symptoms are considered.

The vaginal canal is to be kept clean and dry by packing it moderately with sublimate gauze, which is to be changed every few days. The patient maintains the recumbent posture for at least two or three weeks, while the bowels are kept emptied by artificial means during the first few days.

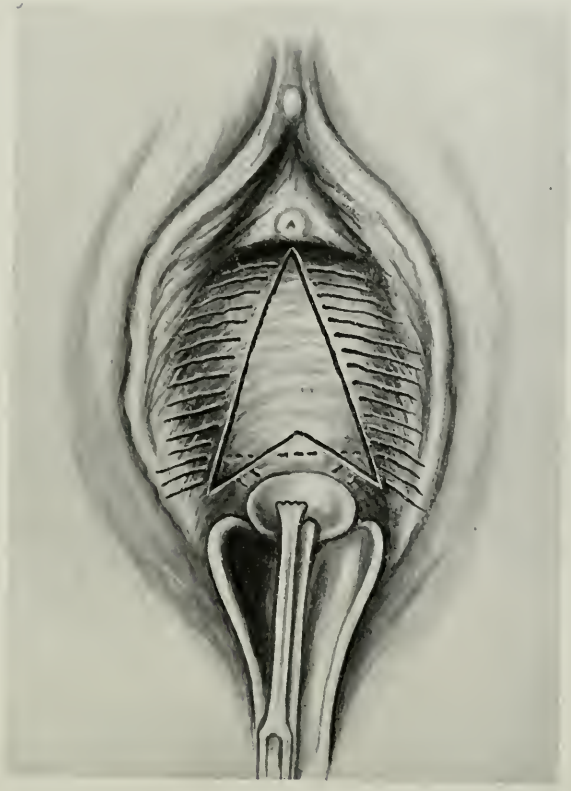
The anterior colporrhaphy surgical procedures have been many, much varied, and for the most part strikingly unsatisfactory. The operation was first proposed by Stoltz. Any one who has performed the operation according to his method must realize that while it narrows the vaginal canal from side to side, it likewise shortens it—a highly objectionable result. Its circular area of denudation with

Fig. 1.



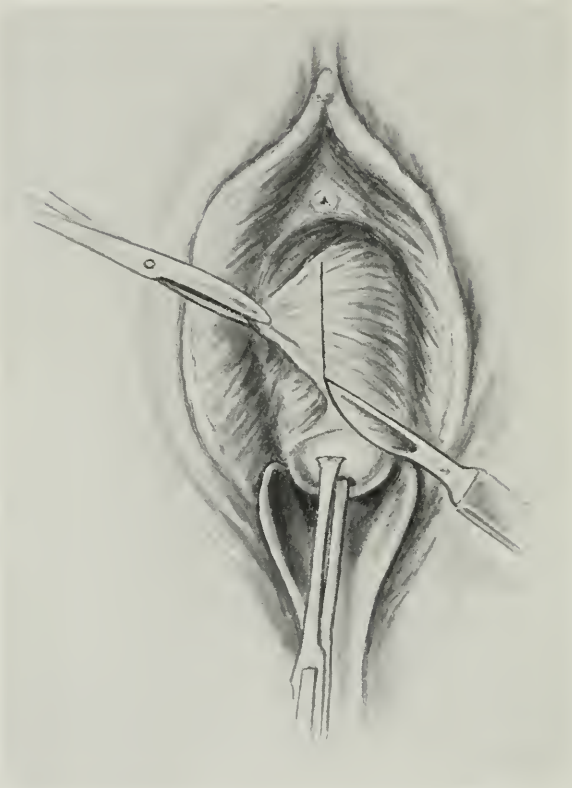
Oval denudation.

Fig. 2.



Shape and technique of Reamy's method.

Fig. 3.



Method of denudation (*Gilliam*).

the purse-string attachments has for years been abandoned for the oval-shaped tract with transverse suturing.

Many years ago, before his decline in health, Dr. Reamy, of Cincinnati, with a full appreciation of the inefficiency of many of these operations, suggested and practised a procedure (Fig. 2) wherein a superior and two lateral areas of denuding were to be made on the right and left lateral surfaces of the vagina about the cervix, to make more sure that the anterior vaginal wall was firmly contracted and that greater upward traction should be secured.

When we recall that the vaginal canal is wider and more baggy above than below we can estimate the worth of his efforts in this direction. As the structures involved in the mechanism of this condition are thin, elastic, weak, and mobile, we can understand why so many cystocele operations are uncertain in their results. The thin, the weak, the elastic, and the mobile must be removed more than seemingly is required, as deep and as wide as it is safe to go and not injure the bladder, and as long as the whole anterior vaginal wall.

Dr. Montgomery, of Philadelphia, has had the same principle in mind. So has Dr. Dudley, of Chicago.

Now Dr. Tod Gilliam, of Columbus, Ohio, comes forward with some further suggestions, especially in the process of denuding, the shape of the area so needed, and the thoroughness and the expedition of his work. He lifts up the redundant tissue of the cystocele with tissue forceps and makes an incision at one side of and near to the base of this fold, in the median line, extending from within a half inch of one end of the fold to within a half inch of the other end. The same incision is carried through the fascia of the muscularis of the bladder (Fig. 3). By a blunt dissection, either with the handle of a knife, the scissors, or the finger, the bladder is peeled, as it were, from the vaginal attachments, after which a suturing transversely effaces the cystocele. The site of operation is then strengthened by an additional layer of fascia taken from the sides of the denuded vaginal surfaces. This method seems highly advantageous in cases in which the cystocele is covered with a large amount of baggy and flabby mucous structure, as in very old cases.

In cases of pronounced prolapsus uteri and procidentia, with of

course large subsequent cystoceles and rectoceles, already referred to, it is prudent, in our judgment, to:

First, curette the uterine cavity, if there is a diseased endometrium.

Second, make an Emmet or a Schröder operation on the cervix uteri, if it presents evidences of an old laceration: an eversion, an erosion, and a hyperplasia.

Third, rectify the cystocele by a Montgomery or a Gilliam procedure.

Fourth, remove the retrocele, and repair the torn or stretched vaginal walls and the perineum. Make thus a firm, strong perineum, by the Hegar procedure.

All of these operations can be done at the same sitting. The recumbent posture is maintained for three weeks, when the patient can carefully and gradually return to the erect posture and her work. The probabilities are assuring that the above operations, well done, will hold the uterus in situ and obliterate the cystocele.

The essential underlying feature, then, in the pathology of cystocele, is the impairment of the integrity of the pelvic floor, occurring during parturition. So it follows, first and foremost, that the whole pelvic floor is to be examined, and insomuch as it has been weakened by lacerations or stretchings, thoroughly repaired in all its parts and relations.

THE USE OF THE STEM PESSARY FOR AMENORRHŒA AND DYSMENORRHŒA: STENOSIS AND ULCER ON LABIA: RUBBER GLOVES: LACERATION OF THE CERVIX AND PERINEUM *

BY J. HENRY CARSTENS, M.D.

Professor of Surgical Gynæcology in the Detroit College of Medicine: Gynæcologist to the Harper Hospital; Consulting Physician to the Woman's Hospital, etc., Detroit, Michigan

THE USE OF THE STEM PESSARY FOR AMENORRHŒA AND DYSMENORRHŒA

GENTLEMEN: I have here a case which is suitable for the introduction of a stem pessary. You have heard me say a great deal about this pessary from time to time, and I thought I would show you how it is introduced. It is the simplest kind of a thing. Every general practitioner can introduce it and use it to advantage in these cases and every general practitioner is confronted with cases of this character that are sometimes very distressing. It is the greatest bore for him to treat these patients where they have amenorrhœa or dysmenorrhœa for weeks and months, when they have it year in and year out, and he has given every remedy for that condition which is made by the manufacturing pharmacists of Detroit, Baltimore, and Philadelphia, and they do not afford relief. It is simply a question of mechanics as a general rule, and the practitioner has a poor and small uterus to deal with.

I use a Chamber pessary like the one I now show you (Fig. 1), which has a certain amount of elasticity. If you press it, it goes together (Fig. 2), and then you can easily put it in the introducer and it slips in easily. One great trouble with most pessaries is that they come out. If the uterus is retroverted, the stem is right in the direction of the vaginal canal and slips out. This woman has a very small cervix. She is suffering from an anteflexion; the uterus comes away forward and is stenosed at the internal os, and that has something to do with the dysmenorrhœa which she has had

* A gynæcological clinic given May 20, 1911.

for a long time. Sometimes she has a little discharge. I will curette the cervix, remove these large glands which project and cause a hypersecretion. There is nothing dangerous about this procedure at all. When I have a younger woman to deal with I do not curette, as they are usually nice and clean. This woman is married, but she has never had any disease.

Fig. 1.

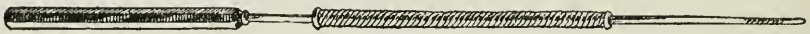
Chamber stem pessary.

Fig. 2.

Chamber pessary, closed ready for introduction

I will now introduce a dilator and open the uterus. This instrument, you see, is an introducer (Fig. 3). I slip the pessary on there, so that the blades will come together, and then I introduce it. For fear that some microbes may get into the cervix and be absorbed by the raw, open channels, I am going to swab the cervix with a little pure carbolic acid before introducing the stem pessary. By that means I close all the raw surfaces because I cauterize them with pure carbolic acid; consequently, if there are any microbes in

Fig. 3.



Introducer.

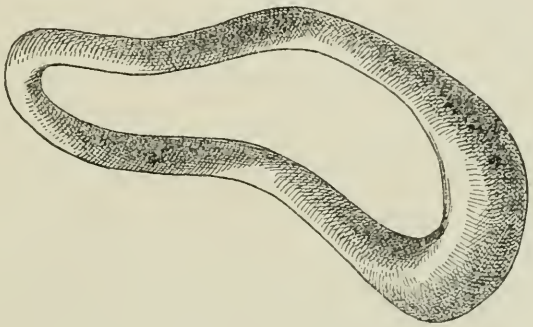
there I kill them and in that way prevent infection. I introduce the stem into the uterus, hold the button there, and I then pull the introducer out. In order to be sure that this button strikes the posterior wall of the vagina, and at a right angle to the axis of the vagina where it is not liable to slip, I put in a Hodge hard rubber pessary (Fig. 4), and put it back into the cul-de-sac; in that way I throw the uterus forward and have this stem right in the centre. You see, I keep the uterus at a right angle to the vagina and the stem always strikes the posterior wall of the vagina and does not come out. By a little practice and experience you get the pessary adjusted in a proper manner.

I saw an article the other day in which an eminent practitioner

of Chicago reviewed the use of the stem pessary. He spoke against the use of this instrument, saying it is dangerous. The stem pessary is not a dangerous thing. I have only had one case where I had trouble with it, and where it caused irritation and had to be taken out; but the diagnosis of absolute confinement to the uterus must be made. If the woman has got a pus-tube and pelvic inflammation, you should not use a stem pessary. It is used, of course, in cases in which there are no adhesions, where the trouble is confined to the uterus, where the tubes and other organs are perfect, but where the uterus is infantile and does not functionate properly.

How do we develop the uterus? Just like anything else that is muscular. How did Johnson develop his muscles? By exercise.

FIG. 4.



Hodge hard rubber pessary.

That is how he got strong and that is how all prize-fighters get strong. The uterus is a strong muscular organ. In these cases of flexion what is the result? Where we have a flexion at the middle these muscles become atrophied and small or the women are born with small uteri or with a small cervix, and gradually the muscle is absorbed and changes and is supplanted by a sort of areolar hyperplasia and fibrous tissue, and that is what causes the trouble and the dysmenorrhœa. In order to keep such a uterus straight, where there is anteflexion, I put in the stem pessary. This stem has branches and they spread out. The uterus tries to contract on it and expel it and cannot do so. The uterus has the power of contraction and always contracts. If there is a foreign body in it, it contracts still more and tries to expel it. This contraction is imperceptible, but it exercises these muscles. Put in a stem pessary

and leave a woman alone for six months or a year and her uterus will become good and straight. She can do anything. This woman can go home. She came from a distance of eight hundred miles to this clinic. She can go home to-morrow without any trouble. She will never know that the pessary is in there; she will be perfectly well; the pessary will interfere with no function whatever, and in the course of four, five, or six months, or even a year, you will see that her uterus has entirely changed. If you resort to a dilatation and curettement in cases like this you will find the woman will be wonderfully well for a short time. The next monthly period will come on just as easily, but in the second monthly period she will commence to have a little pain; in the third monthly period she will be about as bad as she ever was, and in the fourth and fifth she will be just as bad. By the fourth and fifth months you have got to do all the work over again. I have seen that trouble in cases in which dilatation and curettement have been practised three or four times, and there has always been a recurrence of the trouble. If you put in a stem pessary and leave it in there long enough, the condition will not recur and the woman will have no trouble. Dilating the uterus causes pain and you have got to give the patient an anæsthetic to do so. It does not take five minutes to introduce a stem pessary. Anybody can do it. The only thing is that you have to look out that the pessary does not slip out. I have tried to make some experiments along this line. I have used a silver pessary which has more spring, like a piece of steel, and which I am going to give a thorough trial.

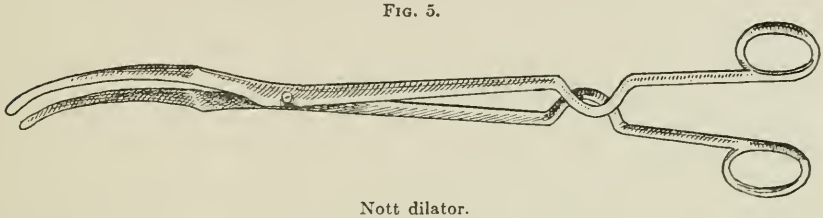
Let me repeat, in conclusion, you must exclude pelvic disease if you would use a stem pessary.

STENOSIS AND ULCER ON LABIA

Here is a patient I have not seen before. She has a very small cervix. There is a little stenosis, but the uterus is fully normal in size; it is large, if anything. She has some trouble at the outlet of the vagina and seems to have some retroversion of the uterus and some discharge. All she needs is a little dilatation. She has no dysmenorrhœa; she menstruates regularly, but has a little pain during the first day. I use this very small Nott dilator (Fig. 5) because the end of it is so small that you can insert it anywhere,

and then I follow it with a Goodell dilator (Fig. 6). She had a little swelling here at the outlet; you can see there is some inflammation present. I was afraid that this girl might have been exposed and become infected, but she is a nice, decent girl. I have had some smears made; I have examined the secretion of the vagina and find

Fig. 5.

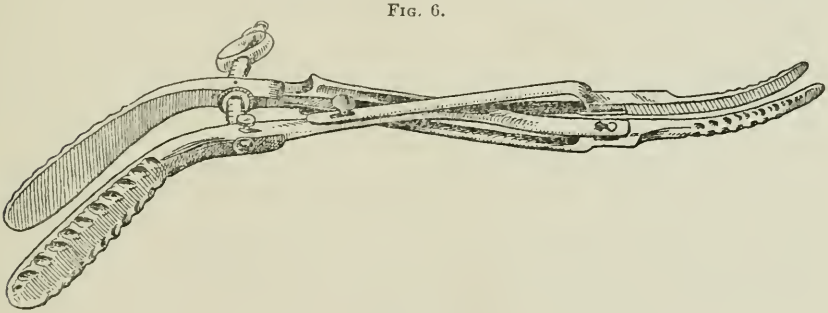


Nott dilator.

that it is simply a staphylococcus infection. We have not been able to find any gonococci in the secretion, so that I know the infection is not gonorrhœal in origin. I simply dilate this young woman's uterus and touch the raw spots with pure carbolic acid. This might have been originally some other condition, some little skin eruption.

I could bring before you cases which would illustrate abdominal

Fig. 6.



Goodell dilator.

operations, but I thought that most of you would rather see these simple cases and how we treat them, so that you can treat similar cases yourselves.

RUBBER GLOVES

In treating this first case you saw that I did not wear any rubber gloves because it was a clean case. In the second case, as I have told you, although I was suspicious it might be gonococcus infection, it was not, still I thought for the sake of safety and to protect my hands I would wear gloves. I practise aseptic surgery

simply by *abstinence from infection*. I keep my hands absolutely clean and never get them contaminated. I go along the street where there is soot and smoke and the soot gets on my hands and your hands and are really black from it, but that soot has passed through the furnace and is absolutely sterile. There is nothing bad about it. You can wash your hands with soap and water and they are clean. Still, for the sake of safety, I wash my hands with all the different kinds of germicides that are in use in order to be sure that they are clean; but I know that I have them clean by abstinence, and if ever I get my hand infected I know that it is going to be infected for at least from four to six weeks. If I ever get my fingers in a pus cavity, no matter how much I may scrape, no matter how many antiseptic solutions of various kinds I may use, I know that my hands cannot be relied upon, and in a certain percentage of cases, those that are susceptible, they will become infected from something on my hands. I may not be able to see it beneath the microscope; I may not catch the spores by cultural methods, but I know that when I never infect my fingers I will not have any trouble. I can then always do clean surgery, but as soon as I get dirty or infected cases, I wear gloves so that my hands will not be contaminated. But I make a mistake sometimes. I operate for a fibroid tumor or something of that kind, for example, and lo and behold there is a pus-tube in connection with it that I was not able to detect. I stop the operation, put on a pair of gloves, and then I proceed to finish it, and if the pus-tube ruptures and I get the pus on my gloves it will not hurt my fingers, but my fingers will be perfectly clean. You may ask the question, Why do you not always wear rubber gloves? Many surgeons do that. Why should I? I can do quicker work without gloves, and so can any surgeon, and I hold that speed counts a good deal. The quicker you can operate, and the shorter time you keep a patient under the influence of an anæsthetic, the better it will be for the patient and the better record you will have. I never made a vaginal examination of any kind without wearing gloves, no matter who the woman is or how clean she is. The vagina is a peculiar thing and it may be infected. I never stick my hands in any pus cavity of any kind without wearing gloves. I am sure that I keep my fingers clean and have the collaterals to prove it.

LACERATION OF THE CERVIX AND PERINEUM

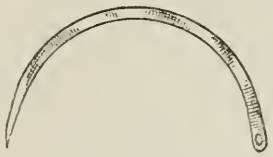
This woman has a lacerated cervix and a certain amount of procidentia. When we have a cervix like this to deal with, with hard nodules, these nodules may be simply from hypertrophy, where the return circulation has been cut off in various directions, more or less, and it is always a suspicious thing, so I have these cases carefully examined microscopically. Now, you can see that this scar goes back to the right side, while the other side seems to be all right and intact. We are not justified in doing anything more than to sew up the cervix and perineum, which we agreed to do. We have no right to take out this patient's uterus, which we might feel like doing. We will denude the surfaces here, and in so doing sometimes I use a knife and sometimes the scissors, and sometimes I use both. We can never tell how small a piece we may find will aid in diagnosis. Thirty or forty years ago you will remember Emmet first taught us that this was a laceration of the cervix, and not an ulcer, and he had much to say about hemorrhages. Everybody got scared and would not dare to undertake the operation. You need not be afraid of hemorrhage. Just think of it, in those days they just got over immense blood-letting. They used to bleed people from sixteen to thirty-two ounces, and then when Emmet came and evolved this operation and cut the circular artery and the woman lost three or four ounces of blood, they were worrying about it, and more particularly if she lost four to eight ounces, whereas a few years before that they used to bleed them a quart at a time. They thought it was good practice then! so I myself never felt the fear of a little lost blood. Of course, we have to use ordinary judgment. In a person who has not very much blood and is anæmic, we want to save all the blood we can, but get down deep enough to get the scar tissue and never mind one or two arteries; pick them up and go on. These operations everybody ought to be able to do.

This woman has a relaxed vagina and her uterus comes down. Various operations have been done on the vagina. I have always made a kind of flap-splitting operation. I never take anything away from the mucous membrane. When a woman has passed the child-bearing period I take away some of the mucous membrane, otherwise I do not do so. In many cases, as in this, I cut at the edge

of the juncture of the mucous membrane with the skin. Sometimes I cut right here in the centre. It does not make much difference so long as you get the parts together. They do not always tear in the middle. They may tear on one side. You can see that these perineal muscles are torn and there is retraction on one side. This one is over on the left side and is retracted an inch or two. Take these ordinary denudation operations and surgeons try to bring the parts together, but we do not get the results, and the principal thing is the pelvic fascia. You cannot get it unless you dissect it out and get hold of it with the forceps and sew it together. If you stick a knife into the perineum or into the vagina it may not cause trouble, but if you stick it into the rectum you are liable to have contamination of the wound, and you had better be careful and not stick it in too far. You can introduce your finger into the rectum and use it as a guide, but in so doing you are liable to have contamination. You get your finger contaminated. At the end of the rectum there are all kinds of micro-organisms, certainly the colon bacilli, so that I do not introduce my finger in there without wearing rubber gloves. We get our landmarks, we render the anterior wall sterile, and we have no trouble at all. In this perineum I find the muscles are pretty well relaxed. These muscles get tired out. Muscles cannot hold up the pelvic organs forever. When a muscle is tired, the layers of fascia hold the muscle in position; they hold the parts in position; by and by the tissues get tired and then the muscles do the work, and so they change about. Now, you see, I bring these together side by side, going away back where the retracted muscles are. You know that there are thirteen muscles in the perineum. I have never tried to remember them all, and I do not ask my students to do it either. I get the perineal fascia and then I get the levator ani and bring them right side by side. Sometimes you have to go over on one side and sometimes on the other. Sometimes you have to put sutures there and sometimes here; you cannot describe it; sometimes you use a running suture and sometimes you interrupt them. You have got to be an artist to do it and describe it. If you are not an artist, try to be as near one as you can. I make use of the same kind of stitch in here as when I have a complete rupture. We have got here the levator ani and we bring these together. I do this operation in various

ways. This is plain sterilized catgut I am using. I do not use any that is chromicized, chemicalized, or anything of that kind. My trouble is in getting them to absorb quickly. Sometimes this suture material will stay and lie dormant for months and months when the patient gets home, and all at once she will get what we call a cold abscess. I take a suture like this, I am using a Hagedorn

Fig. 7.



Hagedorn needle.

needle (Fig. 7), and bring the edges of the skin together. That I simply put in there to hold the skin well together, if there should be an infection or an abscess formed. You can see that it gives a good solid body to the perineum. You can see how we restore the perineum and what the results are. We get hold of the muscles and dovetail them together again, and we have got the perineum restored.

Biography

BENJAMIN RUSH, 1745–1813 *

BY THOMAS W. HARVEY, M.D.
ORANGE, N. J.

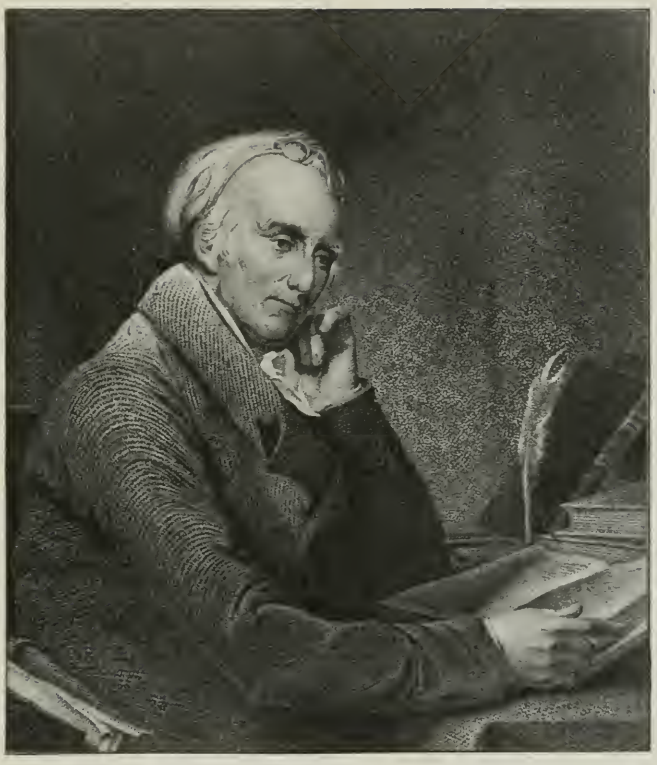
The general catalogue of Princeton College, under the caption of the class of 1760, has the following:

Benjamin Rush, A.M., M.D., LL.D.
Prof. Chemistry College of Philadelphia [afterward to be the University of Pennsylvania], 1769–1789.
Member, Penn. Provincial Congress, 1774.
Surgeon, Penn. Navy, 1775–'76.
Member Continental Congress, 1776–'77.
Signer, Declaration of Independence, 1776.
Surgeon Gen'l, Middle Dep't Continental Army, 1777.
Physician Gen'l, Middle Dep't Continental Army, 1777–'78.
Member, Penn. Constitutional Convention, 1782.
Prof. Theory and Practice of Medicine, U. of P., 1789–'91.
Prof. Clinical Practice, U. of P., 1791–1813.
Prof. Physics, U. of P., 1796–1813.
Founder of Dickerson College.
Treasurer, U. S. Mint, 1797–1813.
Founder and Vice-President, American Philosophical Society.
A.M., Princeton, 1763. M.D., Edinburgh, 1768. LL.D., Yale, 1812.

He graduated from Princeton at the age of fifteen, took his medical degree at twenty-three, was a college professor at twenty-four, signer of the Declaration of Independence at thirty, and Surgeon General and Physician General at thirty-one.

He founded the Philadelphia Dispensary, and attended the Pennsylvania Hospital for thirty years, never missing a daily call, and never being more than ten minutes late. He was also a voluminous writer. In "Allibone's Dictionary of Authors" we find a list of twenty-five titles, only six of which might be called strictly medical, and two of these, "Ardent Spirits" and "Tobacco," were

* Read before the Orange Mountain Medical Society.



DR. BENJAMIN RUSH.

written as much from the point of view of the moralist as of the physician. "The Bible as a Text-book," "The Study of the Classics," "Negro Slavery," "Politics," "Agriculture," "Capital Punishment," are among the many topics on which he wrote and show the wide interest he took in all humanitarian subjects.

Surely we have here to study a life full of interest to the student of medical history; one worthy to be held up as an exemplar for the guidance of medical men, directing them to their opportunities as men, as citizens, and as physicians. Here was a great man and a great opportunity, and he made the most of it. On his return to Philadelphia he plunged directly into public affairs, first busying himself with the young medical school, then taking an active part in the patriotic agitation that led up to the issuing of the Declaration of Independence, and assisting in his professional capacity in organizing the medical department of the Colonial troops. He is said to have severed his connection with military affairs because he found it impossible to stop the frauds perpetrated on the soldiers in the matter of hospital stores, and thenceforth devoted himself assiduously to his profession—teaching, hospital work, and private practice. He served, however, in the Pennsylvania Constitutional Convention, and as a reward for his public services he was appointed treasurer of the United States Mint. He took an active part in civic affairs and wrote much on public morals.

He planned and founded the first dispensary in America, and taught daily in its clinics and in the lecture rooms of the university, having had more than 2250 pupils. His private practice was always very large and he is said often to have treated a hundred patients a day. During the epidemics of yellow fever that prevailed during his lifetime he never forsook his post, even when most of his private patients and many of his colleagues had fled to the country. He was ascetic in his habits, and so devoted to his profession that he would not marry until assured that such a venture would not interfere with it. He was an ardent admirer of General Washington, and was attached to his headquarters during the campaigns in Pennsylvania and New Jersey, but parted from him politically after the government was established, because of differences of political opinions, Rush favoring many reforms, such as the abolition of slavery, far in advance of the public opinion of the day.

Dr. Rush was a man of calm exterior, a philosophical student of disease, willing to change his expressed opinion in the light of new discoveries, beloved by his students, and admired and honored by most of his contemporaries as few men have been. He was so sensitive to criticism that he seriously contemplated leaving Philadelphia, where he lived and worked all his life, because of it.

To the student of medical history the greatest interest is found in the rise and fall of so-called medical schools, or better schisms. Perhaps we may feel that our enlightenment protects us from such vagaries and that medicine is soon to present a united front against the assaults of quackery and fraud. If, however, we look dispassionately over the field of medicine to-day we will see that we are now witnessing the death struggles of one great schism. For many years it has drawn to itself many devotees and its priesthood has increased and waxed great, and many startling prophecies have been made as to its future, but now the guild of priests is diminishing in numbers, and the curtains are drawn decently about the deathbed, and it is only the faithful devotees kneeling devoutly near that do not know that the "risus sardonicus" is about all that there is left of a once perfectly good schism. In its place, however, we find growing and flaunting themselves two very vigorous schisms which are destined, just as in the case of homœopathy, to drive the few grains of truth which have become mixed up with their theories and the undoubtedly useful methods that they have tried to account for by their theories into the minds of men who in the name of medicine are striving for the truth wherever it may be found, and who are willing to follow the logical deductions of discovery no matter into what strange fields they may lead. Eventually, what is good in osteopathy and Christian science will be absorbed into the great mass of medical knowledge and our successors will have to face new forms of schisms.

Now, if we turn reverently back to the latter part of the eighteenth century we will find that a most influential centre of medical education was at Edinburgh. Here flourished the famous William Cullen, one of those great British physicians who have worked themselves to the highest point in their profession from the deepest poverty, and who are admired for their love of science and their devotion to their art. Dr. Cullen introduced a new theory of medicine, attributing every morbid activity to the influence of

the nervous system, and made a notable classification of diseases. He had as a student a certain John Brown, who, similarly sprung from a low origin, also became famous. He did not follow his master entirely, but like him devised a new theory of medicine. I simply wish to introduce him as the author of a system of medicine which has had much influence on the world and which was being taught in Edinburgh when the subject of our paper was a student in that town.

Previous to Dr. Rush's return to Philadelphia the doctrines of Boerhaave were universally accepted as the basis of medical practice. Boerhaave was a great eclectic, who had built up a system of medicine founded on logical deductions from what was then known of physics, chemistry, and physiology, and which has been designated the humeral pathology. The author was undoubtedly the most renowned physician of the world in his generation.

Rush taught the principles of John Brown, or what he called excitability, as the foundation of disease; differing from him in many important details, however, particularly as to the state of the blood-vessels in fever. To quote, he says: "Dr. Brown supposes a fever to consist in debility. I do not admit debility to be a disease, but place it wholly in morbid excitement invited and fixed by previous debility. He (Dr. Brown) makes a fever to consist in a change only of a natural action of the blood-vessels. I maintain that it consists in a preternatural and convulsive action of the blood-vessels." Rush taught that all kinds of fevers are preceded by general debility, natural or accidental. From this a sudden accumulation of excitability takes place whereby a predisposition to fever is induced. Further, *all* diseases are preceded by debility. There is but one exciting cause of fever, and that is a stimulus which depends on a preternatural and convulsive action of the blood-vessels. In another place he says: "All fevers have but one approximate cause and that is morbid excitement, the seat of this is in the arterial system." There are thirty-seven different states of fever.

He did not believe in the nosology of Cullen; fortunately his idea of its unimportance did not prevail, and Cullen's classification has been the framework that has supported our medical knowledge down to the era of the germ theory of disease. He (Dr. Rush) said that names did not amount to anything, that there was one fever, but many varieties, and that the minute classification of

Cullen confused the mind of the student, preventing him from studying the nature of disease. He held that the medical student should use judgment and observation instead of reading and memory, which sounds very much like the criticism we hear to-day about the modern medical school.

In a recent oration on John Hunter and his museum, Mr. Edmund Owen points out how often John Hunter anticipated the discoveries of Pasteur and Lister. For instance, lecturing on hectic fever one hundred years before Lister, he talks about pus and putrefaction, ascribes hectic fever to the absorption of pus, and says that it should be treated by antiseptics. Pursuing a similar method in regard to Benjamin Rush, we will find germinating, sometimes even fully developed, in his teeming brain many ideas and observations that have in later years become our most important and valuable discoveries and agents for human advancement, not only in medicine, but also in many other humanitarian phases of life.

He was an ardent advocate of the abolition of slavery, and shortly after the Revolution wrote a paper on the subject that portrayed the sorrows and persecutions of the enslaved from a humanitarian standpoint. This paper antedated by many years the work of Garrison and Mrs. Stowe.

In the organization of the new nation, in which he took an active part, he advocated the appointment of a cabinet officer who should be the Secretary of State for Peace, his duties to be the encouragement of all things that would tend to make men peaceful; anticipating Carnegie by over one hundred years. He had some quaint views about the methods to be used for this purpose, methods which clearly showed his knowledge of human vanity. He urged the doing away with military clothes and military titles, thus taking away the principal attractions of militarism and incentives to war.

He urged the more thorough education of women at a time when there was very little provision for training them beyond the three R's.

He was one of the earliest to oppose public punishment for crime, claiming that the effect of publicity was not to inculcate fear of the law but sympathy for its victim. Similarly he opposed capital punishment, claiming that his horror of murder in any form caused him to object to the judicial form.

A believer in the classics, he anticipated the elective system of

the modern university, and insisted that too much time was spent on the study of the dead languages and that more practical studies should be used in the training of the young.

He was a most earnest advocate of temperance, preaching strongly against the use of the more fiery liquors, and advising the use of beers and light wines if alcoholic stimulants must be used. He considered drunkenness to be a disease and his description of the effects of acute alcoholic poisoning is a classic.

"The effects of ardent spirits divide themselves into such as are of a prompt and such as are of a chronic nature. The former discover themselves in drunkenness, and the latter, in a numerous train of diseases and vices of the body and mind.

"1. I shall begin by briefly describing their prompt, or immediate effects, in a fit of drunkenness.

"This odious disease (for by that name it should be called) appears with more or less of the following symptoms, and most commonly in the order in which I shall enumerate them:

"1. Unusual garrulity.

"2. Unusual silence.

"3. Captiousness, and a disposition to quarrel.

"4. Uncommon good humor, and an insipid simpering or laugh.

"5. Profane swearing and cursing.

"6. A disclosure of their own on other people's secrets.

"7. A rude disposition to tell those persons in company whom they know, their faults.

"8. Certain immodest actions. I am sorry to say, this sign of the first stage of drunkenness sometimes appears in women, who when sober are uniformly remarkable for chaste and decent manners.

"9. A clipping of words.

"10. Fighting; a black eye or a swelled nose often mark this grade of drunkenness.

"11. Certain extravagant acts which indicate a temporary fit of madness. These are singing, hallooing, roaring, imitating the noises of brute animals, jumping, tearing of clothes, dancing naked, breaking glass and china, and dashing other articles of household furniture upon the ground, or floor. After a while the paroxysm of drunkenness is completely formed. The face now becomes flushed; the eyes project, and are somewhat watery; winking is less frequent than is natural; the under lip is protruded; the head inclines a lit-

tle to one shoulder; the jaw falls; belching and hiccup take place; the limbs totter; the whole body staggers. The unfortunate subject of this history next falls on his seat; he looks around him with a vacant countenance, and mutters inarticulate sounds to himself. He attempts to rise and walk; in this attempt he falls upon his side, from which he gradually turns upon his back. He now closes his eyes, and falls into a profound sleep, frequently attended with snoring, and profuse sweats, and sometimes with such a relaxation of the muscles which confine the bladder and the lower bowels as to produce a symptom which delicacy forbids me to mention. In this condition, he often lies from ten, twelve, and twenty-four hours, to two, three, four, and five days, an object of pity and disgust to his family and friends. His recovery from this fit of intoxication is marked with several peculiar appearances. He opens his eyes, and closes them again; he gapes and stretches his limbs; he then coughs and pukes; his voice is hoarse; he rises with difficulty and staggers to a chair; his eyes resemble balls of fire; his hands tremble; he loathes the sight of food; he calls for a glass of spirits to compose his stomach; now and then he emits a deep fetched sigh, or groan, from a transient twinge of conscience, but he more frequently scolds, and curses everything around him. In this state of languor and stupidity he remains for two or three days, before he is able to resume his former habits of business and conversation."

Discoursing further on this subject, he says that the Pythagorean doctrine of the transmigration of the souls of animals and men was possibly intended only to convey a lively idea of the changes which are induced in the body and mind of man by a fit of drunkenness: "In folly he resembles a calf, in stupidity an ass, in roaring a mad bull, in quarrelling and fighting a dog, in cruelty a tiger, in fetor a skunk, in filthiness a hog, and in obscenity a he-goat."

However, after a long discussion of the subject he philosophically says: "I despair of being able to call the votaries of Bacchus from their bottle and shall therefore leave them to be roused by the more eloquent twinges of the Gout."

He taught that consumption is best treated by exercise and employments that give the greatest vigor to the constitution, urging outdoor sleeping, long journeys over land that involved out-of-door life, long sea voyages, and change from sedentary to out-of-door occupations. He says: "The system should be raised to the high-

est degree of the scale; nothing short of free and vigorous action of every muscle and viscus of the body will fully bring the system up to the point where a radical cure of the disease is possible." He recognized the possibility of a radical cure one hundred years ago. He recommended that patients should seek high and dry air, and should live in a room easily and thoroughly ventilated.

He treated cholera infantum with fresh air, change to country life, the cold bath, and cleanliness in surroundings, teachings which cannot be improved on to-day. The only addition that the men of our generation have made has been clean milk.

As a military surgeon he observed the great fatality among soldiers from disease, and urged improvements in camp and personal hygiene. He calls attention to the fact that in ancient Greek and Roman military annals we have no account of diseases affecting troops to the extent that was observed during the Spanish War, in which of nine thousand deaths occurring in the British Army, seven thousand were caused by fever. Dr. Rush attributes this difference to the fact that the ancient campaigns did not involve much change of climate, and that the use of the arms and weapons of the ancients induced a higher standard of health among the soldiery than do gunpowder and musketry. He considered that the camp and personal hygiene of the Roman armies was calculated to increase the resisting power of the soldier.

In studying the cause of yellow fever he was most active in urging the non-contagious nature of the disease, and curiously noted the constant association of swarms of mosquitoes and insect pests during the prevalence of the pestilence.

He called attention to the character of accouchements among the Indians, "where the woman simply bathes herself and returns in a few days to her usual employment. So that she knows nothing of those accidents which proceed from the carelessness or ill management of midwives." Here recognizing a source of puerperal diseases so urgently insisted on by Holmes and Semmelweis fifty years later.

As a student, in preparing his graduation thesis on the subject of stomach digestion, he experimented upon the acidity of stomach digestion by means of a test-meal and an emetic.

This prevision is the attribute of genius, and we can certainly class Dr. Rush among the great geniuses of the world, and he suf-

fered in his day and generation from the same treatment that has often been meted out to others of his kind who have ventured to observe things through their own eyes rather than through orthodox spectacles, and who have believed that truth should prevail though the heavens fall.

Dr. Rush was a very acute observer in disease. In one place he remarks that many things are true in medicine, as in other branches of philosophy, which are very improbable. While he had very little respect for the *vis medicatrix naturæ* of Sydenham, he nevertheless recognized that often Nature was successful in combating disease unaided by the skill of the medical man, illustrating his argument by quoting instances that he had observed in his study of the diseases of the North American Indian.

Among civilized races, however, he points out the many ways wherein Nature fails: "Being too feeble in malignant fevers and too strong in certain conditions, as in cholera morbus. Being entirely idle in gout, in cancer, in epilepsy, in venereal diseases, apoplexy, and in tetanus, while she does mischief by wasting her efforts in unnecessary fever in consumption or a dropsy, and by terminating a simple serous pleurisy in an empyema.

"Again, Nature often misleads when she does not warn the system early enough by pains, as in anæmia, scirrhus, or stone, or by indications not proportioned to the danger, as in tetanus, consumption, and hydrocephalus, or (oppositely) by too much pain, as in paronychia and toothache."

He points out the effect of civilization in hiding the frank characteristics of a disease and then quotes a colleague approvingly who foretells truly that "gout will be lost in a train of hypochondriacism, hysteria, and bilious diseases."

He makes another observation that is characteristic of his acuteness and his independence: "The signs and symptoms of disease are so modified by climate, habits, and natural characteristics that one may not argue from one country to another. I honour the name of Hippocrates; but forgive me! ye votaries of antiquity! if I attempt to pluck a few grey hairs from his venerable head. I was ever an idolater at his altar, nor did I turn apostate from his worship, till I was taught that not one tenth part of his prognostics correspond with modern experience or observation."

Dr. Rush was certain that you could not deduce from the effects

of treatment on the anæmic and degenerate denizens of London any indication for the treatment of the sturdy and rugged inhabitants of the new world, and he claimed that it was foolish to treat them with the supporting treatment of his quondam teacher, Dr. Brown, but that these sons of the pioneers would stand, and needed much more active treatment, and therefore he insisted on the efficacy of bloodletting and purgation.

Always a follower of the French physician Botallus, called Sangrado because of his fondness for the lancet, Dr. Rush was so pleased with his results in the yellow fever epidemic of 1790, when he commenced his treatment with "10 and 10," 10 grains of calomel and 10 grains of jalap, following that dose with a venesection to 10 or 12 ounces, a procedure to be repeated day after day if necessary, that he adopted this method of treatment as specific for yellow fever and urged its use in all cases.

Those were terrible epidemics that visited our country during the last ten years of the eighteenth century. In the summer of 1790 in Philadelphia there were 4144 cases, and the disease was very fatal. In spite of the attacks of many of his medical brethren and of anonymous pamphleteers, his success in caring for the victims of this disease even by such violent measures was so much greater than that of his colleagues that he soon became the most noted physician in America. And his "10 and 10" was sold over the counter as a specific, much to his dismay.

He published a treatise on the epidemic of 1793 which is one of the most notable medical papers that we have. Sir Benjamin Richardson calls it a most fascinating book. It was widely read in Europe and was quoted as an authority by every writer on yellow fever down to the beginning of the work leading up to the establishment of the mosquito theory.

Dr. Rush's fondness for the lancet was not confined to yellow fever, in which disease he often bled eight or nine times, taking from 50 to 150 ounces of blood altogether, but he used it freely in pleurisies and pneumonias, and, in fact, in almost all conditions accompanied by fever.

I quote from the opening chapter of a manuscript copy of notes of a course of lectures by Dr. Rush on "Pathology, Symptoms, and Treatment," the following. It illustrates his philosophical mind

and the deep religious feeling with which he studied the phenomena of disease.

"We are now to consider living man as subject to sickness and death, the natural consequences of moral evil, and of the loss of primeval innocence. In this condition every element in nature conspired against man. The earth and the sea, the air, aliments and drinks, insects and even his pleasures all seem to aid the offended majesty of Heaven in destroying or curtailing the limits of human life. Their action at first, however, was slow and feeble, and some men attained to the age of almost a thousand years; and it was not until after the Deluge that the term of human life was curtailed to the present limited period. Many are the causes to which its present brevity are ascribed, among which are the Deluge and its consequent effects.

"The idea that life is a forced state and the effect of impressions is as consistent with religion as it is with true philosophy, as life consists in a strife or temporary victory over the causes which tend to produce death. Let us not suppose that the Creator delights in the misery of his creatures. So far from it, all diseases are blessings in disguise, they are necessary to our general good and happiness, their uses are of the first importance, they lead us to the study of subjects highly important to the human race.

"1st. Diseases render it necessary for man to undertake the study of anatomy. 2nd. They promote physiological knowledge. 3rd. They lead us to the study of nature in the animal, vegetable and mineral kingdoms without which the works of nature would remain by us unexplained and unadmired, and without which we would remain ignorant of chemistry and botany. 4th. They lead us to the important study of the human mind and furnish us with frequent opportunities for improvement. 5th. They afford numerous exercises for our moral faculties; for without disease there would be no emotion of benevolence, no acts of charity, nor should we see hospitals established for the relief of the indigent and distressed. 6th. As darkness gives charm to light, in like manner is disease necessary to impart a relish for health. 7th. We should not leave this world without regret were there no diseases. 8th. They reconcile us to the pain, sufferings, and death of our dearest friends and sometimes make us rejoice in the moment of their deliverance. 9th. By their physical effect on the moral faculties they

promote virtue. How many owe their virtue to a fit of sickness? The virtue thus created is of a passive nature which is far superior to active virtue. 10th. Pain alone has numerous advantages. It perhaps contributes to the support of life, it tends to impart vigor to both mind and body, it is the forerunner of disease and tends to point out its seat, it prevents intemperance and frequently operates on the cure of disease. In general, those diseases are most fatal which occur without pain."

Great events breed great men. The momentous period of the American Revolution was fruitful in soldiers and statesmen, the kind of men most needed to meet the requirements of the hour; but there was evidently great need for a high-class medical mind, independent and ready to cut loose from the traditions of European medicine, quick to observe the changed conditions in the New World, quick to note the different effects of old methods of treatment and quick to adapt the old and invent new methods, to meet the indications caused by a new climate, a new life, and new diseases. To meet the need came Benjamin Rush, the American Sydenham, as he has been called. Interested in all forms of altruistic and humanitarian work; impatient in the presence of all "old fogyism" and intolerance; devoted to his profession, not only on its scientific side, being an ardent student, carrying always with him his note-book in which to record his observations of the phenomena of disease and the deductions to be made from his observations, but also on its humane side never tiring when work was to be done and thanking God at the end of the day that "I have seen one hundred patients to-day and none of them have died." I can finish this short tribute to his memory no better than by quoting the opening sentences of his book on "Diseases of the Mind," written the year before his death, which gives a very clear idea of the reverent mind with which he approached his medical studies:

"In entering upon the subject of the following inquiries and observations I feel as if I were about to tread upon consecrated ground. I am aware of its difficulty and importance and I thus humbly implore that Being, whose government extends to the thoughts of all His creatures, so to direct mine in this arduous undertaking that nothing hurtful to my fellow-citizens may fall from my pen, and that this work may be the means of lessening a portion of some of the greatest evils of human life."

Economics of Medicine

THE AID ASSOCIATION OF THE PHILADELPHIA COUNTY MEDICAL SOCIETY

BY OSCAR H. ALLIS, M.D.

Sometime Surgeon to the Presbyterian Hospital; Chairman of the Committee on Benevolence of the Aid Association of the Philadelphia County Medical Society, etc., Philadelphia

Thirty-four years ago the Charter of the above-named association was obtained from the Philadelphia courts and a philanthropic work set in motion that is unique in many particulars.

One of the prime movers in creating such an institution was Prof. Henry H. Smith, long the honored occupant of the Chair of Surgery in the Medical Department of the University of Pennsylvania, and the president of the County Medical Society. It would, indeed, be a difficult matter to point out the most illustrious presiding officer of that county society, one of the oldest and most flourishing in the country, but no one could write its history and not refer to the epoch-making influence exerted upon it by the personality of Henry H. Smith. As a presiding officer he was never at a loss for the very word he needed, and during his many successive terms in office not a single decision of the Chair was ever called in question.

Professor Smith had seen new life spring up within the society; the sparsely-filled benches became, under his popular administration, too few for those who regularly attended, and what could have been more natural than for him to feel that such a society, composed of many of the most active, intelligent, and influential members of the profession, should take up so important a matter as looking after the widows and orphan children of such of its number as should be unable to make provision for them?

The writer, with many others, took no interest in the subject, and it is quite probable that he and many others, would have needed no other incentive to cut a meeting than the announcement that the subject of an aid association would be brought up.

A score or more of years passed by, when the writer, lying upon a sick-bed, too critically ill to be able to turn from side to side, received in his mail one morning a certificate informing him that he had been made a life member of the Aid Association of the Philadelphia County Medical Society.

It was not that he needed it; it was not that in case of his death the society would befriend those whom he would leave behind that touched him—for none of these exigencies could have arisen—but the sick-bed is a keen searcher of hearts and affords many long hours for consideration of topics that are brushed aside in times of health and prosperity.

On recovering his health one of the first acts of the writer was to send his check for one hundred and five dollars to the treasurer of the society, and enter upon such duties as the society should see fit to impose upon him.

At the time of his entrance Professor Smith was there to welcome new members, and his cordial welcome to the writer has never been forgotten. But it was plain to be seen from the actions—not the words—of the directors that the results had been very disappointing. Its organizers had felt that with few exceptions the great majority of the Medical Society would see the necessity and advantages of such an organization and be prompt to avail themselves of it. Instead of the eagerness anticipated, the very suggestion of such a society seemed repellent, and new members were only obtained by personal solicitation—and these too few, too scattering, and at too long intervals to give much encouragement to the few who were laboring in its interests.

It was from Dr. Dunmire, one of the early treasurers, that the writer learned that Professor Smith, sadly disappointed in the success of the organization, proposed to his associates the advantages of separation from the County Medical Society and becoming an independent organization. His action did not, however, meet with approval, and, learning this, he acquiesced in their wishes and labored for its success to the end of his life.

But possibly this portion of the discouraging evil days of the aid association will not interest the general reader who may be desirous of knowing the design of the founders and what the society has accomplished. But these details are gone into to show others who

may be inclined to start similar associations that they must not be pessimistic about its final results.

The second item of the Charter reads:

> "The object of the said association is benevolent, and the purpose for which it is formed is to afford aid to its members and their families, and, in extraordinary cases, to other needy physicians or their families."

The society to-day has one hundred and two members out of a medical society closely approaching fifteen hundred. We congratulate ourselves and feel we have had a prosperous year if a half dozen new members are received and several thousand dollars added to its permanent fund, which is now growing at the rate of nearly five thousand dollars a year.

The organization was designed primarily to help its members, but experience has shown that the benevolently disposed who join the society have no need for its assistance, and join it with no other motive than to help raise a fund that may be drawn upon when distress invades the home of any physician making him powerless to defend and protect his family. As to the membership, fifty-eight have died since the society was organized, but only about one-half of one per cent. have required aid during their lives or their families after death.

The charter includes "other needy physicians and their families," and experience shows that over seventy per cent. of the beneficiaries have been of this class, with no claim upon the society, and who must have suffered had there been no aid association to come to their relief.

Some of the work of the association will no doubt interest the general reader.

A physician, graduate of the medical department of the University of Pennsylvania, found in his early work that he could spend some of his time in reading medical proof at a large printing concern. As time wore on, more of his hours were given to this, and he finally gave up all thoughts of practising his profession—devoting all his time to proofreading. But a fire burned down the establishment where he was employed—one of the largest medical printing houses in this city—he was thrown out of work, and ill-health rendered him an object of charity. The Aid's attention was

called to his case. The Committee of Benevolence paid him a visit, paid one-half his board at the Presbyterian Hospital—the Hospital trustees kindly sharing in the benevolent work—and this man was kept until he was well.

A physician wounded his hand in his office. For one year he was unable to attend to work, and at the end of this time it was necessary for him to be sent to a hospital. An appeal came to the Aid bearing half the entrance fee in each case.

Two physicians who had outlived their earning days, had outlived their families, and had become burdens upon friends who could ill afford to care for them, have been helped to get a comfortable home in that noble charity, The Old Man's Home, the Aid bearing half the entrance fee in each case.

Last summer word came that the widow of a physician was an inmate of one of our hospitals with incurable disease. One of the officers of the society visited her and left her one hundred dollars, and by a recent action of the directors she will be cared for during the remainder of her life.

Poverty is hard enough when it confronts one in the vigor of manhood. Poverty is oppressive and tyrannical when it assails one in the decline of life with all the avenues of self-help barred and closed. But when poverty, old age, and pitiless disease combine, then despair would seem the only refuge.

These cases could be multiplied, but enough have been cited to show the object and working of the society. The reader must not forget that none of the cases mentioned had any claim upon the society, and herein lies the peculiarity, the distinctive character of the Aid. What other charitable organization helps any but its members? If there is one it is unknown to the writer. There is another feature of the Aid that is quite as significant as the one just mentioned, and that is that there is no permanent record kept of its benefactions. The Chairman of the Committee of Benevolence gets a personal receipt whenever he gives any money to one in distress, but the order for the money upon the treasurer is a *numeral:* it may be X or XX. The Chairman has the check cashed and pays out the money, and every year the Treasurer and Secretary compare the vouchers of the Committee of Benevolence with the books of the treasurer, and if found correct all the names of

Beneficiaries and annuitants for the year are torn off and destroyed.

The Aid has shown in its past history that it is worthy of confidence. All the moneys donated go into a general fund which is put at interest for the benefit of such as require aid. The society pays no office rent; no officer of the society receives a salary, but the society, with almost miserly economy, husbands all its resources, now amounting to over $36,000, for the unfortunate, and for them only.

There is a need for such a society as this in every county in every State in the Union. What the future of this society will be cannot now be anticipated. That it will live and in a modest, unostentatious way dispense its benefactions is as certain as any human undertaking can be said to be certain. Whether it will remain distinct as an aid association or be merged with a more notable undertaking in the form of a Home for Physicians is a question for the future. There are few modes of disposing of wealth comparable with looking after those who have, through age or misfortune, become dependencies. All the churches are alive to this great subject, and it ill becomes the army of physicians in this one of the oldest and most charitable cities in America to leave the unfortunate in their ranks without protection in their declining years.

BY JOHN AUER, S.B., M.D.

Associate Member of the Rockefeller Institute, New York City

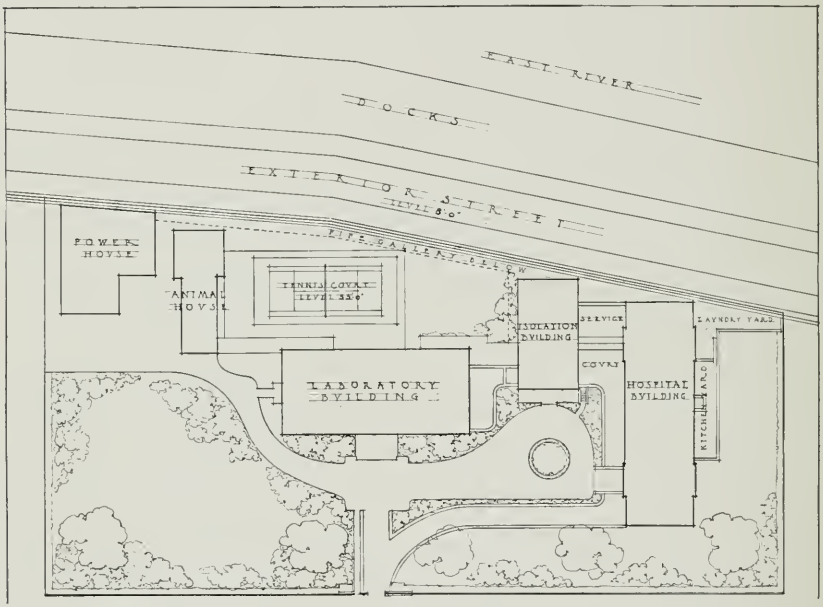
DEVELOPMENT

Up to 1900 there was no institution in this country devoted entirely to research in medicine, but in 1901 this lack was remedied by Mr. John D. Rockefeller. In that year the legal incorporation of such an institute was rendered possible by the pledge of $200,000 from the founder of this Institute; with this sum of money the expenses of ten years' scientific work were to be met. In order that productive work should commence as early as possible, the Institute began at once to aid research by creating money grants and distributing them to qualified workers both here and abroad. After a year's trial it became evident that still better results might be obtained if most of the Institute's work were carried out in one place. After careful investigation New York City was chosen and an adequate site purchased and deeded to the Institute by Mr. Rockefeller. While plans for the final home of the Institute were maturing, several years passed. In 1904 a small building at 127 East Fiftieth Street was leased and equipped with the essentials for laboratory work. In this building, the first home of the Institute, investigations were begun under the directorship of Dr. Simon Flexner. In these temporary quarters research work in experimental pathology, physiology and

chemistry was carried on for two years, until in 1906 the final home of the Institute was ready for occupancy.

THE INSTITUTE

The Institute is composed at present of a Laboratory Building, a Hospital, an Animal House, and a Power House. All are situated on a rocky bluff overlooking the East River between Sixty-fifth and Sixty-sixth streets, and are surrounded by a large free space.



THE LABORATORY BUILDING

THE LABORATORY BUILDING

The laboratory building is a fire-proof structure of steel frame, with outer walls of yellowish-gray brick and limestone. The facade shows five full stories, and a basement which is half above ground. The accompanying plans give an accurate picture of the subdivision of floor space. In general, it may be said that the basement contains some of the machinery of the building and the heavier laboratory apparatus, such as steam evaporation basins, larger centrifuges, shaking apparatus, etc.; the store-room, including a large cold-

VIEW OF THE HOSPITAL FROM THE EAST RIVER

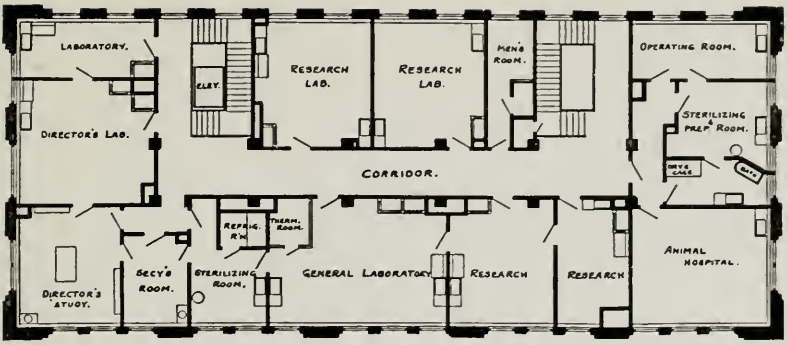
storage chamber for the safe-keeping of various chemicals; a room for the older files of periodicals; the janitor's quarters, and equipment for carpenters' work. The first floor contains the administration offices, the director's room, the library, and an assembly room. The

assembly room is used for society meetings and for the weekly conferences of the Institute staff.

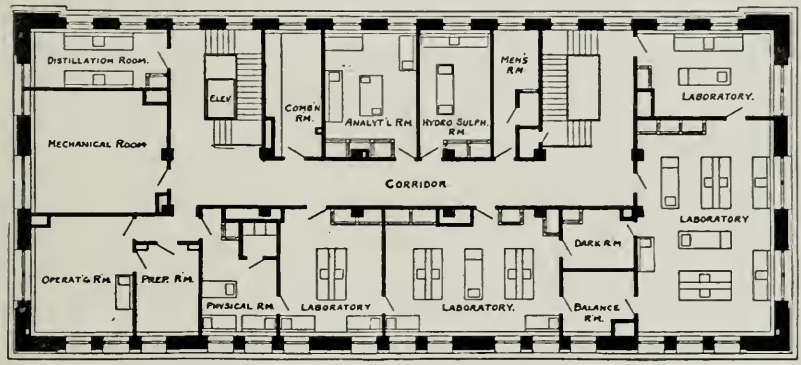
The second floor is occupied entirely by chemical laboratories. On the third floor are large and small laboratories for experimental pathology, bacteriology, and protozoology; at the north end of this floor are the director's private study and laboratory; the south end

OPERATING ROOM IN THE LABORATORY BUILDING

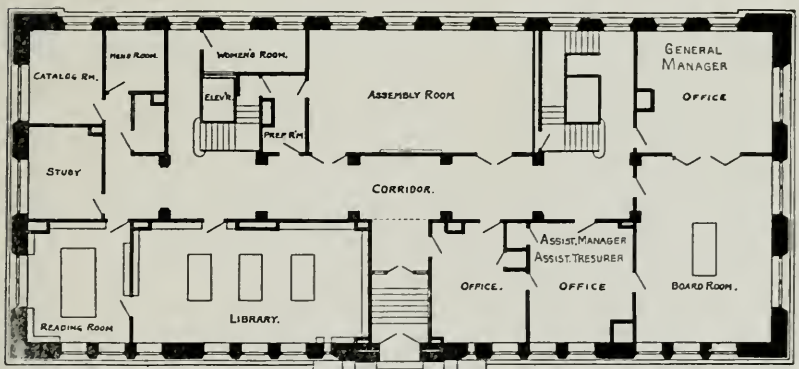
of the floor space is occupied by a series of rooms for aseptic surgical work and by a room paved with concrete in which the smaller laboratory animals may be conveniently kept while under observation. On the fourth floor are laboratories of varying size for experimental pathology and for physiology and pharmacology; there are also rooms for the preparation of media and for the smaller centrifuges.



THIRD FLOOR PLAN



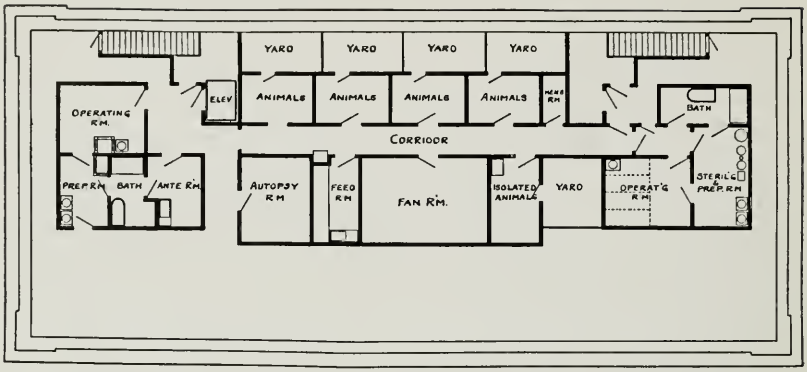
SECOND FLOOR PLAN



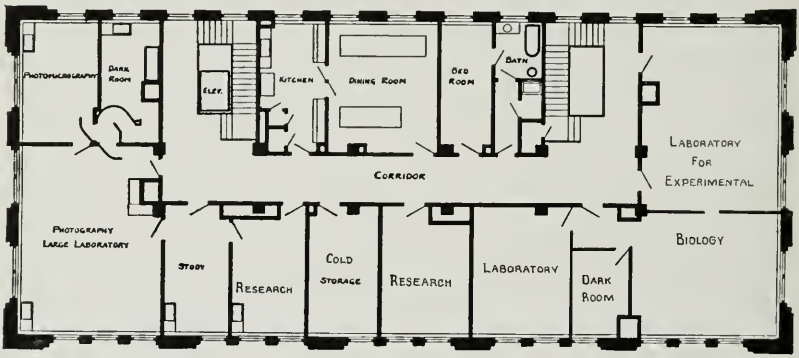
LABORATORY BUILDING
THE ROCKEFELLER INSTITUTE FOR MEDICAL RESEARCH

FIRST FLOOR PLAN SCALE 0 5 10 15

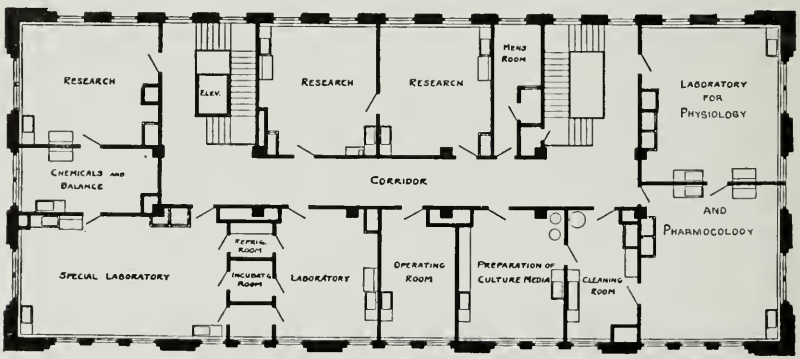
SHEPLEY RUTAN & COOLIDGE ARCHITECTS



ROOF PLAN



FIFTH FLOOR PLAN



FOURTH FLOOR PLAN

LABORATORY BUILDING

SHEPLEY, RUTAN AND COOLIDGE, ARCHITECTS

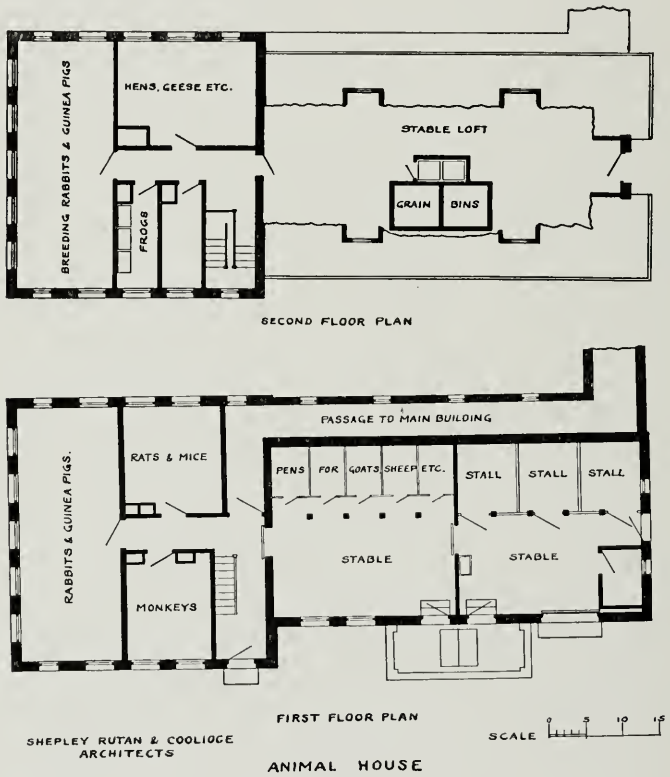
The fifth floor contains the laboratory rooms devoted to experimental biology; rooms equipped for photography and for the illustration of publications; a few special research rooms, and a kitchen and two dining rooms for the scientific and clerical staff. The roof, a large part of which is covered over, is subdivided into a number of small rooms for the housing of dogs. Each one of these small rooms is provided with

CHEMICAL LABORATORY IN THE LABORATORY BUILDING

concrete floors and an outside tiled runway. At the north and south ends of the roof there is a suite of operating rooms; the southern suite has been especially arranged for this work and its north wall and roof are constructed of glass, so as to furnish the maximum amount of light with a minimum of interfering shadows.

All the laboratories are fully equipped with everything needful

for the particular work which is being carried on. On each floor there is a large ice-box reaching from floor to roof, which communicates with the main refrigeration plant in the power house and with a subsidiary refrigeration plant in the basement. The various floors of the building are connected by two stairways, one at each end of the building; in addition there is an electric elevator. The building is fully wired for local and long distance connections, and on each floor there is a telephone booth.



ANIMAL HOUSE

North of the Laboratory Building and connected with it by a covered passageway is a two-story semi-fireproof structure for the housing of the larger laboratory animals, such as goats, sheep, and horses. The stables for these animals are on the first floor. On the second floor are large, well-lighted, airy spaces where monkeys, cats,

BUILDINGS AND GROUNDS OF THE ROCKEFELLER INSTITUTE. (VIEWED FROM AVENUE A.)

rabbits, guinea pigs, mice, and various species of fowl are kept. A small room containing a series of tanks is utilized for the storage of frogs. There is also adequate provision for the storage of grain and hay.

An important adjunct to the animal house is a farm, located in New Jersey. This place furnishes a considerable fraction of the fodder necessary for the animals and provides a place where the smaller animals are bred. In addition it serves as the summer home of animals which have been operated.

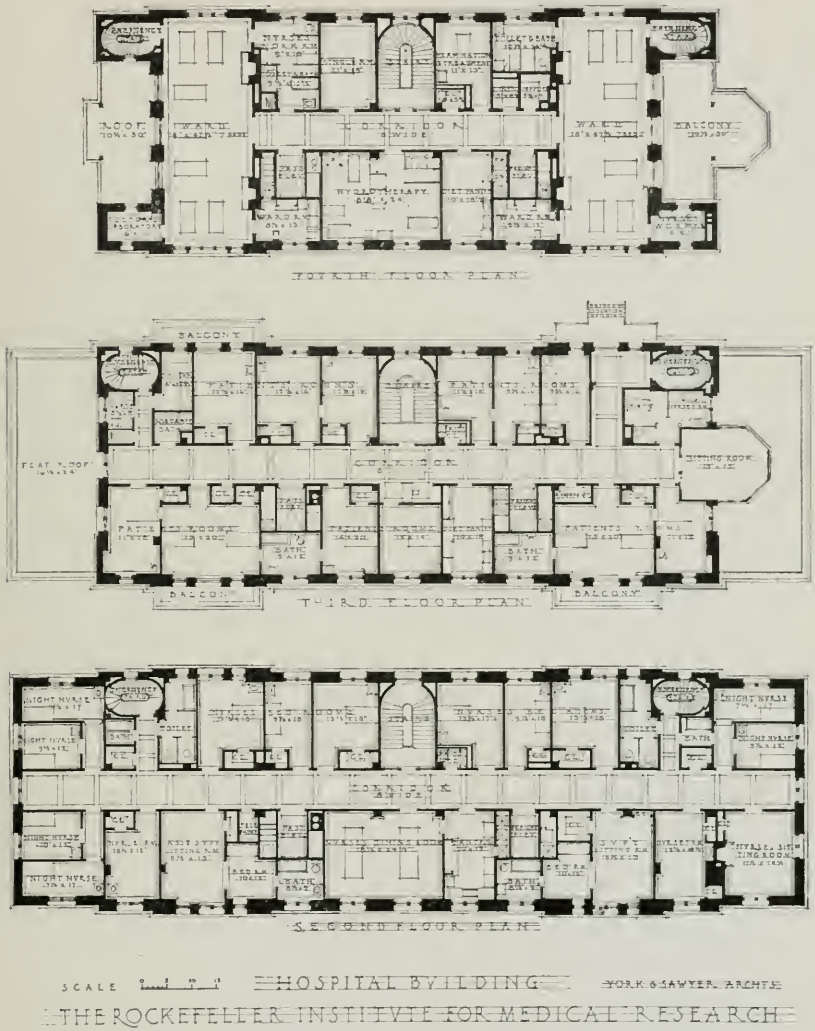
PART OF THE LAUNDRY

POWER PLANT

All the steam heat, electric light and power, and refrigeration used by the various buildings of the Institute are furnished by the power house, which is located north of the animal house. The equipment includes four 60 H.P. return tubular boilers; three side-crank engines, driving one 100-kilowatt and two 50-kilowatt direct-current generators; one twenty-five-ton compression type refrigerating machine; one three-cylinder motor-driven vacuum pump; a fully equipped motor-driven machine shop, and a complete laundry.

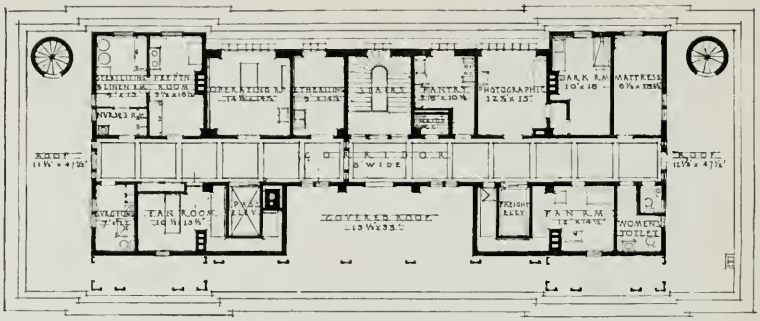
THE HOSPITAL

The hospital was completed in October, 1910, and consists of a Main Building and an Isolation Pavilion. The buildings are fire-

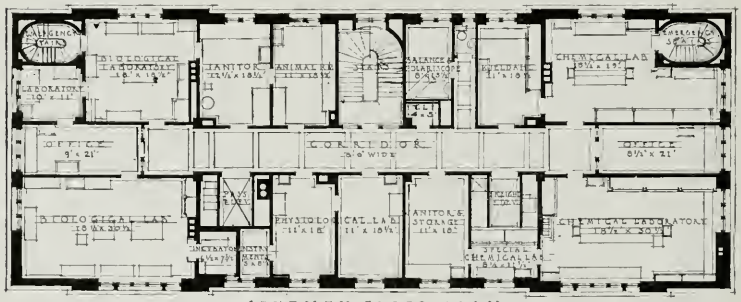


proof throughout, being constructed of steel, concrete, brick, and terra-cotta.

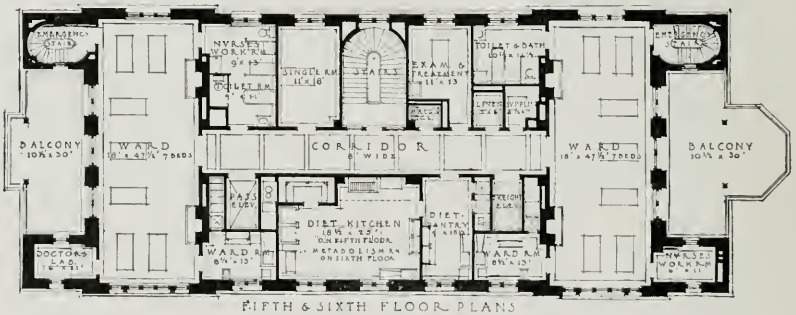
The main building shows a pleasing facade of artistic brick-work in its upper stories, the first two stories being sheathed with limestone. There are eleven stories counting three basement floors



EIGHTH FLOOR PLAN



SEVENTH FLOOR PLAN



FIFTH & SIXTH FLOOR PLANS

SCALE: 0 5 10 15

HOSPITAL BUILDING

YORK & SAWYER, ARCHTS.

THE ROCKEFELLER INSTITUTE FOR MEDICAL RESEARCH

and a covered roof. A clear conception of the subdivision of the various floors and their use will be obtained from the floor plans of the hospital accompanying this article. It will be observed that the

fourth, fifth and sixth floors are identical in plan, and these floors largely determine the plan of the entire building. At each end of these floors is a ward running north and south, at right angles to the long axis of the building, which gives a maximum amount of light and air to these wards. The space between the wards is taken up by the ward kitchen, toilet, and work rooms; there are also two separate

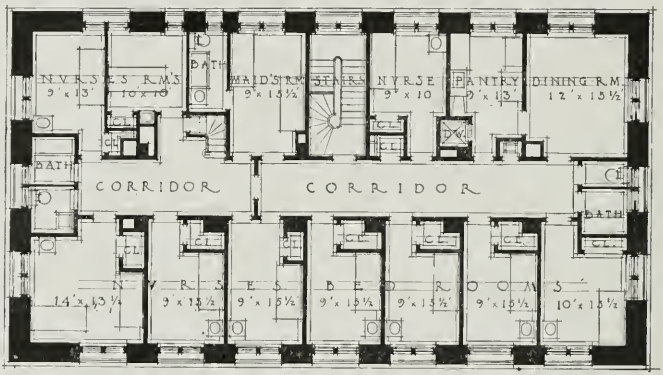
A WARD IN THE HOSPITAL

rooms each designed for the accommodation of a single patient. The wards are all eighteen feet wide and forty-eight feet long and each contains seven beds. Each ward opens in a balcony large enough to hold all the beds at one time.

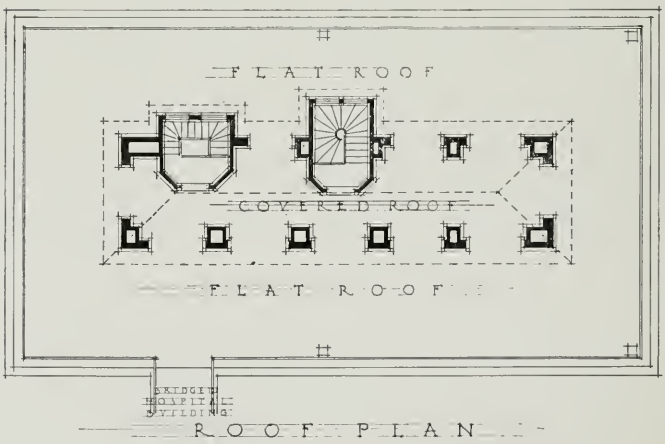
The seventh floor is arranged entirely for laboratory work in pathology, chemistry, and physiology. There is the usual equipment,

and in addition an animal room and a constant temperature vault. A constant temperature room for experiments in metabolism is located on the sixth floor.

On the eighth floor there is among other rooms a small operating suite for emergency use, and a photographic and X-ray suite. At the northeast and northwest corners of the building are enclosed fire-escape stairs which connect all the floors and lead from the ground to the roof. The main stairways are cut off on every floor by fireproof screens and doors.



SECOND FLOOR PLAN.



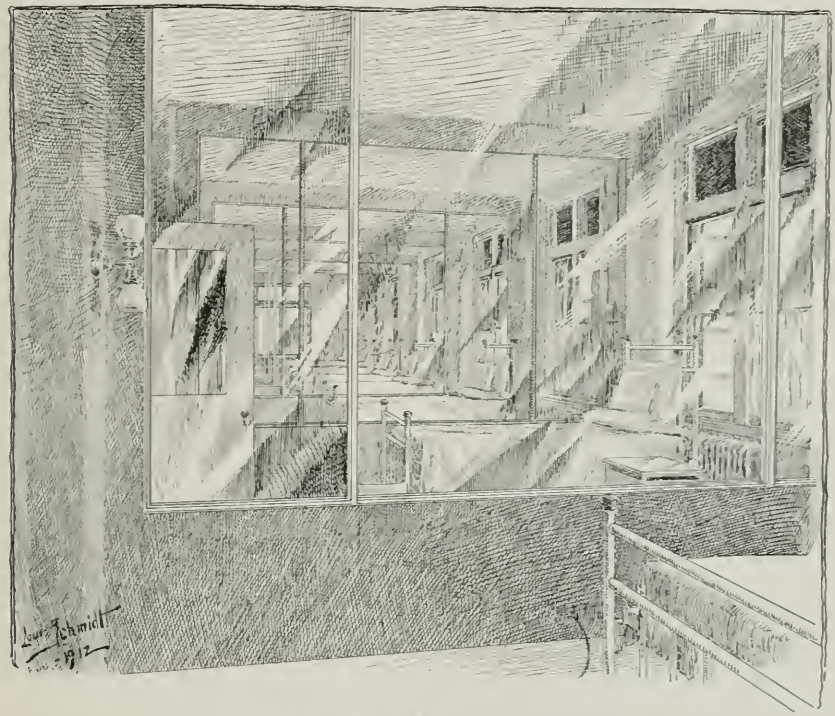
ROOF PLAN.

ISOLATION BUILDING.

The ventilation in the wards and single rooms depends chiefly upon the windows and doors. There are two electric elevators, one for passengers, the other for freight.

ISOLATION PAVILION

This Pavilion is located between the laboratory building and the main building of the hospital. The main floor forms practically a single ward, as the partitions are of glass; this facilitates the super-



WARD IN THE ISOLATION PAVILION

vision of patients and removes largely the irksome sense of solitary confinement experienced by convalescents. The intake of fresh air depends upon the windows, but ventilation is aided by flues carrying steam-pipes which run from each room to the roof. These flues can be sterilized by jets of live steam. The second floor is occupied entirely by nurses. The roof is partly covered over with a copper sheathing.

The work of the Hospital is confined to the study of selected types

MR. JOHN DAVISON ROCKEFELLER. (FROM THE BUST BY MR. WILLIAM COWPER.)

of disease, and during the first year the diseases chosen were acute lobar pneumonia, acute anterior poliomyelitis, syphilis, and certain forms of cardiac disease. The services of the Hospital are free, and no charge of any kind can be made under the by-laws of the corporation. The capacity of the Hospital is about seventy beds.

ORGANIZATION

Departments of the Institute.—The laboratories of the Institute are under the general directorship of Dr. Simon Flexner and their scientific activity is subdivided into the following departments:

(1) *Experimental Pathology.*—This department is the largest and its activities the most varied, for it includes pathology, bacteriology, protozoology, and experimental surgery. This department is under the direction of Dr. Simon Flexner and his staff, formed by Drs. Noguchi, Carrel, Rous, Terry, Clark, Lamar, Manwaring, Murphy, Wollstein, Bronfenbrenner, Butterfield, and Tytler.

(2) *Physiology and Pharmacology.*—This department is directed by Dr. S. J. Meltzer. The staff consists of Drs. Auer, Joseph, Kleiner, and Githens.

(3) The *Department of Chemistry* is in charge of Dr. P. A. Levene, and his staff is composed of Drs. Heinrod, Jacobs, Van Slyke, Birchard, Courtney, La Forge, and Meyer.

(4) *Experimental Biology.*—This department is under the direction of Dr. Jacques Loeb, and his staff is formed by Drs. Bancroft, Wasteneys, and Beutner.

The Hospital.—The medical work of the Hospital is in charge of its Director, Dr. Rufus Cole. He is assisted by a staff consisting of the Resident Physician and a number of Assistant Resident Physicians. The clinical staff is aided by special workers in chemistry, pathology, bacteriology, and physiology. Dr. Cole's staff is as follows: Drs. Robinson, Cohn, Dochez, Draper, Ellis, Peabody, Swift, and Gillespie, and Miss Fales.

ADMINISTRATION

The Board of Trustees has the custody and management of the endowment funds and other properties of the Institute, and it possesses the power to choose directors. This Board is composed of Messrs. Starr J. Murphy, Simon Flexner, John D. Rockefeller, Jr.,

Frederick T. Gates, and William H. Welch. The endowment funds amount to about $7,186,000.

The scientific policies of the Institute are determined by the Board of Scientific Directors formed by Dr. William H. Welch, Dr. Theobald Smith, Dr. T. Mitchell Prudden, Dr. Simon Flexner, Dr. L. Emmett Holt, Dr. Hermann M. Biggs, and Dr. Theodore C. Janeway. This Board decides the appointments to the scientific staff,

A CHEMICAL LABORATORY IN THE HOSPITAL

their rank and stipend, and the assignment of grants to qualified extra-mural workers.

APPOINTMENTS

Appointments to the staff of the Institute are made by the Scientific Board of Directors when recommended by the Director of the Laboratories or the Director of the Hospital. These appointments, as a rule, are made only as vacancies occur, but application may be made at any time. Members of the Staff ordinarily devote all their time to research and there is no provision for the instruction of regular classes of students.

The grades of the Institute Staff are as follows: Member of the Institute; Associate Member of the Institute; Associate; Assistant; Fellow; Scholar. The medical staff have additional titles: Physician to the Hospital; Assistant Physician to the Hospital; Resident Physician; and Assistant Resident Physician. Appointments of Members are made without any time limit; Associate Members are appointed for a number of years, and the other appointments are for a term not exceeding one year.

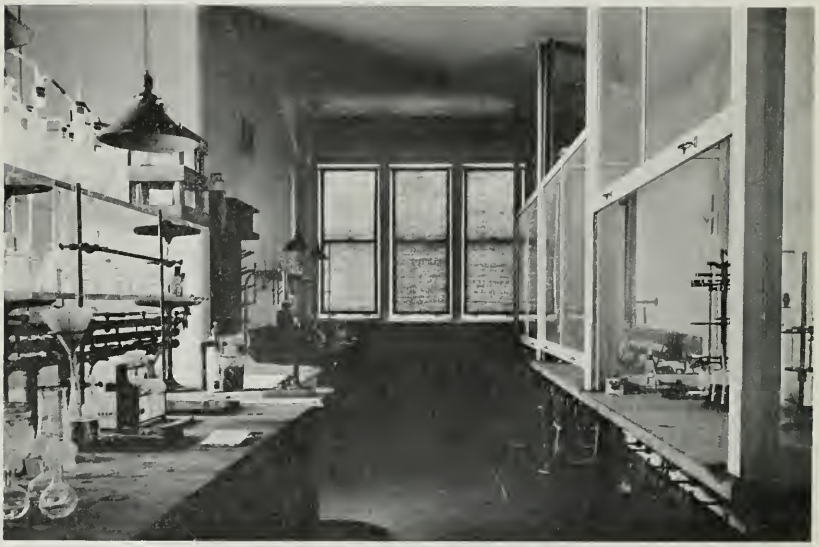
GRANTS

A number of investigations aided by grants are carried on outside of the walls of the Institute. The amount of money available for this purpose is limited and determined each year by the Board of Scientific Directors. This aid to extra-mural workers is extended under certain conditions, which may be learned upon application to the General Manager, Mr. Jerome D. Greene.

CHARACTER OF THE WORK OF THE INSTITUTE

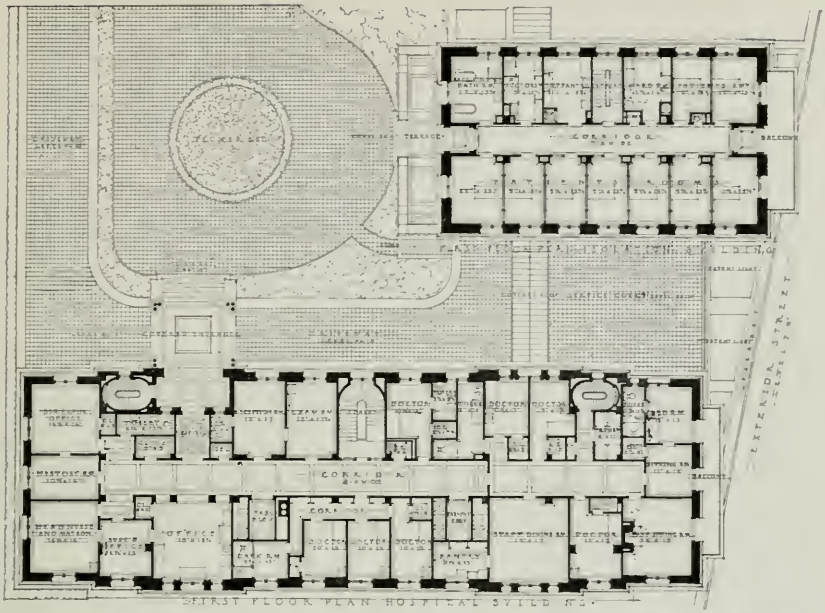
The Institute was founded and incorporated for the purpose of furthering research in medicine in every possible way. This does not mean that only the so-called practical problems are investigated, though a considerable part of the energy of several departments is devoted to this end. This happens because the activities of these laboratories are more closely related to practical medicine than others, but it by no means follows that these results only are of value to medicine. Even a casual consideration shows that all knowledge gained in the biological sciences must in the long run be of service to humanity, for the fundamental manifestations of life must be the same in the lowest as well as in the highest forms, there is a conservation of specific function, in other words, and the existing differences can only be those of degree and specialization. All possible information regarding functional manifestations are therefore of great value to medicine, as their counterpart must exist in man. But another aspect appears when we consider that all disease is merely an exaggeration, either in a minus or a plus direction, of the normal every-day functions of the organism, for obviously disease can only manifest itself by disturbing existing functions, it cannot create new ones; the creation *de novo* of functions is no more possible than

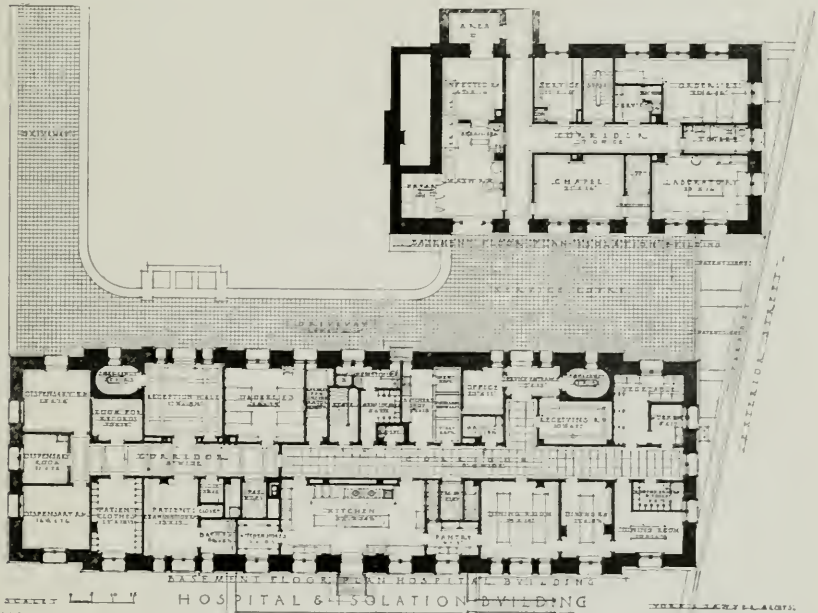
the creation of new matter. Since all disease may be looked upon as a distortion of the normal processes of life, and since all information regarding normal functions thus necessarily gives information about disease, the vast importance of all biological knowledge becomes obvious even to one who tests the results of these investigations by the question, "What's the use of it?" There is, however, a point to which attention may be called. The results obtained in pure science are, as a rule, not immediately applicable to the needs of man; results obtained in the biological sciences, for example, must usually be



A CHEMICAL LABORATORY IN THE HOSPITAL

modified and fitted, so to speak, to the necessities of the human subject. The average biologist is not able to do this because he lacks the necessary medical knowledge, and the average practitioner cannot do it because the biological law is often so general that it means nothing in particular to him. The attempt of either may lead to misunderstanding: the scientist listens with amazement to the scientific explanation of a disease process made by the practitioner, and the practitioner stands aghast at some of the practical suggestions offered by the medically ignorant biologist. It was this condition, in my opinion, which drew the sharp line of demarcation between the


FIRST FLOOR PLAN HOSPITAL BUILDING
HOSPITAL & ISOLATION BUILDING
THE ROCKEFELLER INSTITUTE FOR MEDICAL RESEARCH


BASEMENT FLOOR PLAN HOSPITAL BUILDING
HOSPITAL & ISOLATION BUILDING
THE ROCKEFELLER INSTITUTE FOR MEDICAL RESEARCH

devotees of the Science and the practitioners of the Art, a separation which was still more accentuated by the conception of scientists that "practical" results meant a commercialization of Science. The two camps, however, are gradually drawing more closely together, due partly to the growing realization that practical results do not necessarily imply a prostitution of Science; that pure scientists are

MAIN ROOM OF THE LIBRARY

not necessarily intolerant and useless visionaries; and partly because a new group of workers has been formed who are qualified to take a scientific view of practical medical problems or a medical view of biological investigations. Such a partnership the Rockefeller Institute is utilizing, as is shown by its scientific organization as well as by the character of its work. The Institute is doing its utmost to investigate disease processes with all the intellectual tools at present available, but it also aids by producing new tools, new biological facts,

to be employed in medicine as soon as they have been fully tested in the laboratory and their practical use seems warranted.

RELATION OF DEPARTMENTS

There is no one specific problem which all the laboratories of the Institute investigate; each department studies those scientific aspects which interest it particularly. There is free personal intercourse between the members of the various laboratories which is aided by various devices. The most efficient is the Institute dining room, where the workers meet one another daily at luncheon. These daily meetings have drawn the entire staff into a relationship, and have produced a sense of unity which probably could not be obtained in any other way. Another aid in promoting acquaintance is a weekly informal conference where designated members present either results of their own investigations, or a review of the literature on some subject. Two papers only are given during these one-hour meetings. A tennis court has also proved of value in bringing the members of the various laboratories together.

PUBLICATIONS

The results of investigations carried out under the auspices of the Institute are published in the journal devoted to the particular field to which the researches belong. A considerable percentage of these papers, however, appear in the official periodical of the Institute, *The Journal of Experimental Medicine.* Long scientific papers are published as *Monographs of the Rockefeller Institute.* At irregular intervals all the published investigations of the Institute, including those published under its grants, are collected and bound into volumes. Up to date, thirteen of these volumes have appeared. Most of these studies are distributed free to the leading investigators and laboratories throughout the world.

SOME RESULTS OF VALUE TO MEDICINE

In a previous section it was pointed out that all biological facts are of value to medicine and that they only differ in respect to their immediate applicability to the problems of practical medicine. Mindful of this fact, it is perhaps legitimate in this place to mention some results obtained by the Institute which are of interest to the practitioner.

Antimeningitis Serum.—This was the first therapeutically active

serum available in epidemic meningitis and has been extensively employed with excellent results both here and abroad.

Intraspinal Injections of Magnesium Sulphate for Tetanus.—This method brings about relaxation and prevents convulsions of the patient when all other methods, including the anti-serum, fail.

Intratracheal Insufflation.—This method gives a ready and safe access to the thoracic cavity for surgical intervention. The method is also used for ordinary anæsthesia, and for all cases of respiratory failure.

There are also numerous investigations whose results promise to benefit practical medicine. The study of infantile paralysis has yielded important information regarding prophylaxis; blood-vessel and organ transplantations have been carried out successfully in dog and cat; a serum for influenza meningitis has been produced which yielded good results in monkeys; the two main causes for acute death in anaphylaxis have been established in animals and the treatment for one type found; the *Spirochæte pallida* was isolated in pure culture and a skin reaction described which permits the diagnosis of the late, masked forms of syphilis when the ordinary tests often fail; studies in tuberculous meningitis have shown that the life of animals suffering from this disease could be considerably prolonged; a new method has been devised by means of which the laity are enabled to give artificial respiration in cases which demand this help.

That the work of the Rockefeller Institute is appreciated abroad is well shown by the fact that Dr. Simon Flexner delivered the Huxley lecture on October 31, 1912, in the Charing Cross Hospital, and that Alexis Carrel received the Nobel prize in medicine for 1912.

This short and summary view of the practical work does not pretend to give an adequate picture of this side of the Institute's activity. It is merely presented to show that the Rockefeller Institute for Medical Research, in spite of its youth, has already justified its existence.

Insanity

THE RELATION OF PRISON LIFE TO THE DEVELOPMENT OF INSANITY AMONG PRISONERS*

BY PAUL E. BOWERS, M.D.

Physician in Charge, Indiana Hospital for Insane Criminals, Michigan City, Indiana

THERE is a prevalent idea entertained by those who are not familiar with the mechanism of organic or functional insanities that the mental experiences incident to life in confinement stand in primary causal relationship to the development of mental diseases which are common among prisoners.

Even among physicians who are trained psychiatrists the idea is so firmly implanted that prison life of itself produces insanity that some members of the German school have designated some of the atypical forms of the essential insanities which occur in convicts as prison psychoses.

One alienist, in supporting the theory that prison life produced insanity, advances the following argument: "It is true, imprisonment acts more deleteriously upon the psyche of the criminal by passion, and the accidental criminal, but even the recidivist, who would be expected to feel less keenly the painful loss of freedom, falls a prey to deleterious effects of prison life. The unfavorable hygienic surroundings which are found in most prisons, the scarcity of air exercise, readily prepare the way for a breakdown, even in an habitual criminal. Above all, however, it is the emotional shock and depression which invariably accompany the painful loss of freedom, the loneliness and seclusion which force the prisoner to a raking occupation with his own mind, to a persistent introspection for the future, the remorse for his deed, that play an important rôle in the production of mental disorders."

Before conceding the correctness of this view it will be necessary to study the mechanism of mental diseases which are induced by

* Read before the American Prison Association November 13, 1912.

the above-mentioned factors, the psychology of the criminal, his physical organization, his burden of heredity, his habits, and the penal institutions and their administrations. It is true that the various experiences of life produce reactions of painful or pleasurable nature, depending upon the character of the occurrences, and the psychical organization of the person receiving them, but these reactions, if not pathologic, do not alter or abrogate the individual's usual manner of thinking, feeling, and acting.

Mental traumatism resulting from the disgrace of criminal conviction and imprisonment has been suffered time and again without the development of a mental disorder. Dr. Carlos F. McDonald remarks: "Substantially every individual at some time or other during his life is exposed, in many cases repeatedly, to many of the so-called exciting causes of insanity, both mental and physical, and yet, despite this fact, we find that sanity is the rule, insanity the exception." We shall expect, therefore, that those individuals who develop psychotic symptoms as sequelæ to imprisonment have for the basis of their mental disorders defective or unstable mental substrata.

Kraepelin has stated that mental disorders develop about ten times more frequently in prison than in the ordinary walks of life, but this is not strange when we stop to consider that the crowds which haunt our prisons are largely composed of mentally and physically defective individuals, who find themselves unable to battle with life because of their abnormal organizations. Many of them belong to the ranks of the crank, vagabond, pauper, and prostitutes,—the slag of social evolution.

"Wilmans, in a study of an hundred and twenty-seven vagabonds, found sixty-six cases of dementia præcox." The penal institutions serve as a social clearing-house, so to speak, but not as ætiologic factors for the production of insanity.

During my experience as a member of the staff of the Government Hospital for the Insane I encountered sailors and soldiers who sought military service in an endeavor to escape their imaginary pursuers and persecutors. Those individuals of the dementia præcox constitution, the constitutional inferiors and organic failures who sought military life in response to the call of the *Wanderlust,* found themselves at last properly classified in the Government Hospital for the Insane.

They escaped close observation until becoming identified with the military, which resulted in the disclosure of their defects. Military life did not primarily create their insanities. Army life serves as a means to segregate the fit from the unfit; the same can be said of the prisons, whose populations, in general, are far below in mental status that of the military.

In order to have a proper conception of the relation between the insanity of prisoners and those factors which are alleged to be operative in the production of psychogenetic mental states, we must acquaint ourselves with the forms of mental diseases which are common in penal institutions. To accomplish this, the prisoner's physiological, anatomical, and mental organization must be analyzed, to determine whether or not there exists an underlying defective foundation upon which active mental disturbances can be erected, or whether or not there is latent mental disease lying dormant to be disclosed by some disagreeable mental experiences of prison life.

Anthropology has thus far failed to establish a distinct criminal type, but marks of constitutional inferiority are exceedingly common in prison populations. Among the anatomical defects are to be found malformations of the skull, teeth, and palate, the Darwinian tubercle, Morell ear and prognathism, all of which may be interpreted to mean reversions to more primitive types.

These same evidences of stigmata are, of course, frequently found in normal individuals, but they so often appear in our defective, dependent, and delinquent classes that they may be considered as marks to differentiate the normal from the abnormal, and they at least suggest that the causes which produced them produced corresponding defects in the psyche.

The physiological abnormalities encountered are perversion of the sexual instinct, uncontrollable desires for liquors, migrane, disorders of the nervous system, insensibility to pain, defects of speech, and reduced physiological tension. The psychic stigmata are more clearly defined here: we find exaggerated egotism, eccentricities, ill-balanced mental activities, irritability, inability of continuous application to mental or manual labor, emotional poverty, brutality, and fatalism. Eroticism is shown in 75 per cent. of all tattoo marks. We will find that the habits and heredity of the criminals are the chief elements in the production of their insanities.

A tabulation of 2,681 consecutive admissions to the Indiana

State Prison, where criminals over thirty, and murderers are received, will be studied. I found that out of this number 2,293 prisoners had used alcohol, 83 per cent. of this number had used intoxicating liquors to excess, getting drunk at frequent intervals, 1,362 admitted that they had been infected with gonorrhœa, 476 admitted infection with syphilis, and we can safely surmise that nearly as many more had been infected, but denied their venereal history; 127 admitted the use of narcotics, 1,879 had been previously convicted of criminal offences. This last fact shows a clear mental defect; namely, a lack of inhibitory control.

One hundred and fifty-three had been convicted of rape, 43 had been convicted of sodomy, 46 had been convicted of incest, making a total of 242 convictions for perversions and inversions of the sexual instinct. Pederasty is by no means uncommon among the recidivist and habitual criminals. One hundred and twelve were actively insane at the time of admission, 47 were epileptics, 596 were classified as dull, nearly 50 as feeble-minded of varying degrees, 622 had relatives who were insane, feeble-minded, or epileptic.

After a careful study of these figures, we are not at all surprised at the large numbers of insanities which develop, and are recognized in prison. Before going further the administration of modern prisons will be reviewed. The prisons of to-day, which are conducted on broad humanitarian plans, evolved from careful scientific study and experience, are based on two fundamental principles: First, that an artificial environment should be created in prison which should resemble those conditions existing in normal society; second, to provide such training for the hand and mind as will secure for the prisoner that degree of adaptability which will enable him to live in society without conflict with his environment when his prison sentence is finished.

The idea of retributive justice is no longer the dominant principle that governs our relation with offenders; penology is no longer a mere matter of speculation: it penetrates deeply into the subjects of biology, eugenics, psychology, and sociology, for crime is now recognized as a social disease. Therefore, in our treatment of the criminal we have incorporated in our system of social therapeusis the same general principles which govern our treatment of the sick. The modern prisons have been styled—and rightly, too—"moral

orthopædic institutes," where deformed and wayward feet are straightened out and put in proper paths.

Our penal institutions are sanitarily constructed, properly ventilated and scrupulously clean; wholesome and appetizing food is supplied in abundance and of healthful variety. Few institutions for criminals nowadays are not supplied with circulating libraries of well-selected books, weekly religious services, musical concerts and frequent entertainments, military drills and calisthenics, but better than all these mental diversions afforded the prison inmates is a daily and wholesome occupation which provides usually some small compensation.

Under the régime of prison life, with its healthful food, daily wholesome employment, regular hours for sleep, and medical care, we find that many prisoners who were on the verge of a mental and physical breakdown at the time of admission, owing to their vicious habits of intoxication, prostitution, and drug ingestion, are restored to health.

After considering the characteristics of the criminal and touching briefly on the methods of administration, construction, and sanitary conditions of prisons, we will study the type of prisoner who develops insanity while serving sentence in penal institutions. In conformity with the theory of psychogenesis, we would conclude that those individuals in prison who meet the requirements for the evolution of purely psychogenetic mental diseases would be the ones to develop insanities.

We do not expect the imbecile to experience much sorrow upon the death of his mother, or the low-grade individual to suffer much shock of his conscience on account of his wrongdoing, therefore, we do not expect the morally anæsthetic or the individual of low-grade intelligence to develop mental disorders on account of the painful experiences of remorse and introspection. If the psychogenetic factors were the real causes of insanities in prison, we would expect that the accidental criminal, or the criminal by passion, should develop insanity, for these individuals most clearly resemble the normal individual, or do not differ from him at all; but, on the contrary, the facts do not admit of this argument, for the higher class of criminals who are capable of emotional distress rarely develop insanity, for these individuals possess what the normal individual

possesses, that capacity for adaptation to the environment, and, having a clear conception of their condition as prisoners, they adjust themselves to their surroundings, conform to the rules and regulations peaceably, secure the easy berths in prison, become clerks and foremen, and fill other offices of trust.

Investigation will quickly dissipate the idea that remorse, sorrow, and introspection for misdeeds stand in causal relationship to the development of insanities of convicts. We will, however, find that the lower grades of criminals, incapable of intense suffering, because of their low-grade mentalities and emotional poverty, are the ones to develop active insanities during confinement. If we study their histories we still find that their lives have been one uninterrupted series of conflicts with the law and morals of society. They are morally anæsthetic, brutal in their passions and indifferent to ethics. Occasionally we find an exception to this rule, so far as intellect is concerned, but the emotional poverty in these exceptional cases is ever in evidence. The episodic, psychotic symptoms which are exhibited in prison are but the kaleidoscopic pictures of their criminal constitutionality which has been their heritage, or which they have acquired by evil environments, vicious habits, and low ideal during the formative period of the mind. The mental symptoms which obtain prison are merely the evidence of underlying grave psychophysical pathology.

The insane in prison populations may be divided into two classes: First, those persons who are insane at the time of admission and whose mental disorders have been overlooked by the dispensers of justice. Nearly 5 per cent. of admissions to penal institutions belong to this class.

The methods of administering justice to the offenders of the law are so distorted, entangled, and confused by the technicalities of our legal system that the insane and mentally irresponsible suffer.

Indeterminable amounts of time are spent in securing testimony and evidence relating to the manner, mode, and legal classification of crime, and the criminal himself is forgotten. The courts of justice largely deal with the symptoms of social disease, instead of dealing with the real cause of crime, *i.e.*, the pathology of our social system; and as one of the results of our inadequate methods of dealing with the criminal problem we have cases of senile

dementia, arteriosclerotic dementia, paresis, paranoia, epilepsy, and dementia præcox sent to prison to be punished for crimes which were merely symptomatic expressions occurring in the evolution of their unrecognized and disregarded mental diseases.

The second class is comprised of those who are potentially insane and who have escaped close observation in the outside world, and, when brought in conflict with the law, develop and exhibit their organic defects, and active psychoses come to light. This class is composed of degenerate persons afflicted with the various forms of hysteria, psychasthenia, imbecility, moral imbecility, nervous diseases, and the sexual perversions. Many individuals who have had psychotic symptoms have passed for sane, when at liberty, because their mental symptoms were not recognized, but upon coming to prison these episodic mental states, which had previously escaped detection, are noticed, and frequently these acute exacerbations are charged to prison life. This last class, with its prepared candidates for active mental disease, furnish the majority of our insane criminals.

Regis states that "the insanity of degenerates depends on grave mental infirmity; in the ordinary insane the psychosis is everything. Here it is only a secondary phenomenon, superadded and often episodic. There are two distinct elements to be considered: the vice of organization, and the mental diseases." Three arbitrary divisions of the degenerate psychoses may be made. However, no distinct line of demarcation can be drawn between them. In the acute hallucinatory form we have hallucinations, maniacal outbreaks, general distractibility, pronounced psychomotor activity, mutism and delirious states; a paranoid state is ofttimes observed; delusions of a persecutory nature obtain, the régime of prison discipline is misinterpreted, persecutory ideas are built on the flimsiest and most chimerical experiences, the paranoid ideas become systematized and tenacious, and more or less dangerous tendencies are shown by the patient. The hysterical form of the degenerative psychosis is often exhibited. Here we find the hysterical stigmata, the restriction of the visual fields, confusional states, affective perversions, and convulsive attacks.

The following case was selected to illustrate the type of prisoners who go insane. He belongs to the class of the habitual criminal

and the recidivist, these being found in the most defective strata of criminal society. The life histories of these classes are such that we are almost forced to accept the theory of Lombroso that criminality is a psychosis in itself.

The psychotic episodes which they manifest in prison are not new creations, but are merely fluctuations of their disequilibriated mentalities.

Family History.—White, male, twenty years of age, parents living, father is a drunkard, mother is an epileptic, the patient is the third oldest of eleven children: one sister has tuberculosis, one brother was convicted of burglary, one maternal aunt is in an institution for feeble-minded, another maternal aunt is a prostitute and horse thief. She has served one term in prison.

Present History.—Patient's birth was abnormal, forceps delivery; when one year of age had anterior-poliomyelitis, which left the patient paralyzed in left leg and arm; did not have the usual disease of childhood; learned to talk at three years of age, learned to walk at the age of six; attended school six years, reached the fifth grade. He was always abused and cruelly treated by his mother and brothers; when fifteen years of age he had a serious quarrel with his mother over some alleged cruelty, said he assaulted his mother; after this affair he left home and hoboed about the country for about six months and then returned home. Shortly after his return he developed a psychotic episode which lasted about three weeks; during this period of time he was confused, delusional, homicidal, and violent, was locked in county jail for safe keeping, and steps were taken for his transfer to an insane asylum, but before this measure was carried out his symptoms subsided and he was released from jail. Upon his release he again resumed the life of a hobo, which he lived for four years, until admitted to prison. During his nomadic career he associated with criminals and prostitutes, drank to excess, indulged in venereal excesses, and practised sodomy; he was arrested at least sixty times and was twice convicted of larceny and served jail sentences. In February, 1911, while hoboing in Indiana, he spent all his funds for liquor and debauches, and in order to secure money planned a robbery. He states that many times he heard his hobo friends tell how easy it was to do a "hold up" and secure some easy money. Acting upon these ideas, he entered the house of a gentleman unknown to him and demanded money. His victim offered some resistance to the intrusion, and the patient shot him, killing him instantly.

Physical Examination.—White, male, 5 feet 4⅜ inches. Head, anterior-posterior diameter, 18.5 cm.; transverse diameter, 14.2 cm.; bimastoid, 12 cm.; bimaxillary, 12.2 cm.; height of forehead, 3.9 cm.; length of nose, 3.2 cm.; right ear, 3 cm.; left ear, 3.3 cm.; cephalic index, 77.66 cm.

Head: Cranial type, mesticephalic, marked plagio-cephalic on left side and anteriorly, palate high, sharply arched and narrow, malocclusion of teeth, beard absent.

Body slender, left arm contracted and atrophied, slight contractures of fingers, atrophy of the left leg and contraction of tendo achillis and tendons of toes, right scoliosis lumbar region.

Neurological Examination.—Reflexes normal except for left patellar and tendo achillis, both of which were absent.

Mental Examination.—Consciousness clouded at times, feels as if he were in a dream or a trance. Says that something is wrong with his mind. Has vague idea of persecution; feels obliged to do certain disagreeable things, unable to control his actions, masturbates frequently, ideation limited and at times confused; his conversation is full of criminal slang, memory poor, moral sentiment almost lacking; he believes it is all right to steal if he escapes being caught. Lacks imagination except for the planning of some crime; emotional poverty evident; prefers idleness to profitable employment; accepts his imprisonment with indifference and unconcern.

Judging from this man's defective organization and history, I am of the opinion that he will, in a short course of time, develop more active psychotic symptoms than he displays at present, and if he does, these cannot be properly ascribed to his environment. The prison will merely serve as a mirror for his degenerate nature.

If the limitations of this paper permitted, I could furnish still more proof, from the findings of psychological examinations and the histories of prisoners, that the primary cause of insanity among them is their active predisposition to mental disorders, and not the environmental influences of prison life, which are merely accidental contemporaries.

I feel that I have emphasized certain points which will tend to dissipate the rather prevalent idea that "prison life of itself produces insanity." The burden of proof is with those who support the prison psychotic complex theory, but the preponderance of evidence is against such hypothesis, for the alleged psychogenetic factors—worry, remorse for crime, apprehension, and introspection—exert but little influence on the psyche of the convicts who develop insanities.

The insane criminals are recruited from the ranks of the recidivists, who do not mentally suffer because their neural organizations are below par, and their normal and ethical senses are blunted and poorly developed.

It may be further noted that recoveries from mental diseases among prisoners are quite rare, from the fact that their insanities are developed on fertile and degenerative soil.

This being true, it is rather difficult to accept the theory that prison life of itself produces insanity.

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